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A CRITICAL EXAMINATION OF THE FOUNDATIONS OF THE "RECOIL OF THE VOWEL" THEORY OF THE CAUSE OF THE IMPEDIMENT OF THE SPEECH IN STAMMERING

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For more than a quarter of a century Dr. C. S. Bluemel, psychiatrist, has worked persistently upon an effort to prove a particular theory relating to the etiology of stammering, and still holds tenaciously to his belief in the truth of that theory. The present writer desires to express his appreciation of Dr. Bluemel's long and consistent effort, especially in view of the fact that the theory involved has never been definitely refuted. Also the present writer approves wholeheartedly of Dr. Bluemel's specific purpose to explain the immediate reason for the stammerer's fundamental impediment in producing speech, because explanation of this impediment constitutes the basal requirement for the solution of the complete etiology of the disorder. If this basal problem can be solved the further facts of etiology, although complicated, can be determined with comparative ease and certainty.

Previous to the work of Dr. Bluemel, attempts to solve the etiology of stammering were concerned, primarily, with direct study of the peripheral symptoms of the disorder, and, secondarily, with their cerebral explanation. Much effort to understand the peripheral symptoms as accurately as possible characterized this earlier period. Dr. Bluemel accepted the peripheral symptoms as he understood them, and attempted from a psychiatric, as well as an introspective standpoint (for he himself stammered), to attach them to a definite psychiatric cause. Thus he was not wholly divorced from the earlier psycho-physiologic point of view.

During the period of Dr. Bluemel's work, roughly the past twenty-five years, psychologists and psychiatrists in considerable numbers have taken up the same problem. Close study of peripheral symptoms has been definitely abandoned. Indeed certain of these

authors have questioned whether stammering was specifically a speech disorder at all. Was it not rather some strange manner of functional psychologic disturbance that had a peculiar relation tc the function of speech, compelling these extraordinary peripheral motor disturbances? Dr. Bluemel has been the leader in diverting the effort to understand stammering further and further away from the earlier and, to my mind, much more rational purpose, to appreciate closely the peripheral symptoms of the disorder, and to trace those symptoms precisely to their cerebral origin.

At the very beginning of Dr. Bluemel's presentation of his then new theory he evidently felt that it must have a foundation that should attach the theoretical psychiatric disturbance which he was bent on expounding, to the symptoms of speech abnormality of stammering. It is this baasl requirement of his theory that I propose to examine.

Dr. Bluemel's Theory

Dr. Bluemel builds the foundations of his theoretical conception as to the basal cause of stammering as follows:[1]

He first discusses an old and formerly a much advocated theory with respect to the nature of stammering, as follows: "The theory supposes that stammering is due to a delay in vocalization; in other words, to the stammerer's inability to produce voice. The theory was first promulgated by Dr. Neil Arnott, in 1827, in 'Elements of Physics.' Later the theory was promulgated, probably independently, by Merkel in Germany." He then quotes John Wyllie,[2] who says, "the speaker neglects the laryngeal mechanism"; and, also, "The defect of speech . . . is due to delayed action of the laryngeal or vocal mechanism in attacking the first syllable of words." Dr. Bluemel also quotes Bastian,[3] who explains the same theory by saying that "stammering is due to a want of accord between the action of the laryngeal and oral speech mechanisms. There is mostly a lagging action of the former."

After stating this theory, as above, which definitely places the cause of hesitation on the laryngeal production of the voice of the sound stammered on, whether a vowel or a consonant, Dr. Bluemel endeavors to refute it as follows: "It is easy enough to show that the difficulty of the stammerer is in some way connected with the production of the vowel, and that the consonant is not the obstacle." He offers the following "arguments" in support of that contention:

(1) "The stammerer usually prolongs the consonant for several seconds." Thus it is evident "that he has no difficulty with the consonant, which is prolonged only because the vowel is delayed."

(2) Also "in *stuttering* (the consonant) is repeated only because the succeeding vowel refuses to appear." Since the consonant is

[1]Stammering and Cognate Defects of Speech. Vol. I. The Psychology of Stammering. New York, 1913, Chapter VII, pp. 181–187.

[2]The Disorders of Speech, John Wyllie, pp. 2–3.

[3]Aphasia and Other Speech Defects. H. Charlton Bastian, 1898, p. 58.

repeated, the author argues, "there is no obstruction to its articulation."

Again, (3) "if the stammerer's difficulty lay with the articulation of the consonant he would stammer quite as much at the end of a word as he does at the beginning," and stammering on the ending consonant does not occur.

(4) The author points out that stammering often occurs on the beginning vowel of a word; "therefore the difficulty does not lie with the consonant."[4]

(5) After stating that singing (in which stammering is usually absent) "differs from speech chiefly in the manner in which the vowel is produced, while there is manifestly no radical change in the consonants," the author then abruptly concludes: "from these arguments it is evident that the stammerer's trouble is due to the delay of the vowel." The fact that "the stammerer is unable to produce voice" arises from his inability to produce the succeeding vowel. Then, as if to clinch finally the conception that the stammerer's difficulty could not possibly be due "to a lagging of laryngeal action," the author makes the following statement:

(6) "Unfortunately the (delayed laryngeal action) theory does not take account of all the facts, and the facts that it disregards are sufficient to refute it. If the stammerer's impediment were due solely to his inability to produce voice, then all trace of the impediment should vanish as soon as he begins to whisper, for in whispering no phonation occurs"; and yet, stammering occurs with about equal frequency on whispered as on loud sounds.

The author, repeating what he has before stated, continues: "A second fact shows just as conclusively that the difficulty is not one of vocalization. One often stammers on continuous sonant consonants, and in this case there is continuous production of voice." An especial example of continuity in the production of the *m* sound in this connection is made. "Here there is certainly no delay in the action of the larynx."

Then the final conclusion is reached: "Now, since the stammerer's difficulty is to produce the vowel, and is not to produce *voice* per se, it is evident that his difficulty is to produce the *vowel-color, or voice quality. The stammerer's difficulty is transient auditory amnesia.* He is unable to recall the sound-image of the vowel that he wishes to enunciate. This, then, is the thesis of the present monograph."

"The stammerer is an auditomoteur. He relies for his speech cues upon both kinaesthetic and auditory images. When he stammers in enunciating a word, it is because there is complete failure of the auditory image. His futile struggles with the initial consonant are directed solely by his kinaesthetic imagery, but he cannot pass to the vowel because he cannot recall its sound, its peculiar characteristic quality—in short, the *vowel-color.*"

[4]A further statement of the author as to stammering on the vowel will be quoted later.

In a later book[5] Dr. Bluemel reiterates the above statement of his theory, and restates it in the following words: "The (first) theory is, I believe, substantially correct, though not sufficiently precise and clarified. Loss of sound imagery undoubtedly occurs; but the difficulty is not so much an inability to *recall* the sound, as it is a recoil of the sound from the mind. Amnesia occurs, not because the stammerer waits upon his memory, but because he is confronted with a break in the continuity of consciousness."

Such is the foundation on which is built a still active psychiatric theory as to the cause of the basal impediment of the speech in stammering. Not *direct* psychophysiologic impairment in the action of the mechanism of speech, but an *indirectly* caused hesitation in its action, due to the momentary inability of the stammerer to visualize the sound of the succeeding vowel, accounts for the delay of speech in stammering, is Dr. Bluemel's contention. I propose to examine closely this effort of the author to build a foundation for his theory. The discussion will be carried on in the order adopted by him, as follows:

1. Stammering on the continuous loud consonant.
2. Stuttering.
3. Stammering on beginning vowels.
4. Absence of stammering on the final consonant.
5. The singing voice.
6. Stammering on the whispered sounds.

I. Dr. Bluemel: "*The stammerer usually prolongs the consonant for several seconds.*" *Thus it is evident "that he has no difficulty with the consonant, which is prolonged only because the vowel is delayed.*" An especial example of prolongation of the *m* sound in this connection is offered.

Since an especial statement is made later as to stammering on the whispered sounds, and since the completely stopped oral consonants (*b*, *d*, and *g*) cannot be normally prolonged, the above statement evidently refers only to the continuous loud consonants. What the author says is that stammering on consonants "usually" consists *only* in "prolonging the consonant for several seconds." I have added to his statement, and have italicized, the word "only" in order to make the author's statement clear. No close observer of the symptoms of stammering will agree to this statement. I would say that in more than nine-tenths of the cases of stammering on the continuous consonant the act of stammering does *not* consist *only* of "prolonging the consonant for several seconds." What about complete momentary stoppage, not only of sound production, but also of the movements of the speech machine itself? What about "blocking" on the production of the sound involved, which includes complete stoppage of the speech machine for "several seconds"?

[5] "Mental Aspects of Stammering," Dr. C. S. Bluemel, 1930, page 44.

The "usual" and typical manner of stammering on any sound is the stoppage of the movements of the speech machine, while the musculature continues in action. This may, or may not be followed by production of the sound involved for a shorter, or longer, number of seconds, and for one or more repetitions of both the stoppage and the sound production. The stoppage of movement of the speech mechanism constitutes the primary and fundamental act of stammering, and of this the author says nothing. What the stammerer fears is his inability to produce a certain sound; and it is the stoppage of the speech mechanism in such an attempt that causes the characteristic struggle in the act of stammering.

And yet occasionally, in the mature stammerer only, I believe, stammering does consist only of prolonging the consonant. This prolongation of the consonant, however, in my observation does not occur "without difficulty," but is usually attended with a hesitating struggle. As the more mature stammering child begins to realize the seriousness of his speech disturbance, he starts upon a continuous effort, which lasts through the entire course of his later stammering life, by which he endeavors to prevent, or, if this be impossible, to lessen, or to modify, his act of stammering. Through this characteristic effort at self control each stammerer comes to develop to a greater or lesser degree his own peculiar modifications of his original act, and, in rare cases, this may consist only of prolonging the consonant, or, for that matter, prolonging the stammered vowel as well.

As to prolongation of the *m* sound while stammering, which Dr. Bluemel emphasizes, my memory recalls only one instance in which this phenomenon has regularly occurred. This happened in a highly neurotic stammerer of extreme degree, who, with respect to other sounds, stammered with the usual stoppage of the speech mechanism, and much struggle. While I may have overlooked other instances, I feel certain that such cases are extremely uncommon. And yet one whose stammering on the *m* sound consists of the usual stoppage of the action of the speech mechanism and consequently of the sound itself, may occasionally prolong the *m* sound.

Conclusion.—Dr. Bluemel has made a serious error in observation. Of course, if a student were to confine his study to a single stammerer who did habitually only prolong the continuous loud consonant, he might naturally, although wrongly, conclude that all stammerers did the same. Could this idea possibly explain the author's error in observation? At any rate his statement is based, I believe, on mistaken observation, and, therefore, as an "argument" possesses no value.

Dr. Bluemel: "*The consonant is prolonged only because the vowel is delayed.*"

This is merely the statement of a preconceived theory. In view of the above picture of what stammering really is, and in view of the fact that the author evidently did not understand in a complete sense the symptoms of stammering, his conclusion is meaningless.

II. Dr. Bluemel:

"In stuttering the consonant is repeated only because the succeeding vowel refuses to appear." Since the consonant is repeated, "there is no obstruction to its articulation."

When an automobile stops, then starts forward a bit, and then stops again, one doesn't conclude, because it can go forward for a moment, that there is no "obstruction" to its action, at least without an examination of the mechanism. The speech machine is complicated in structure and action. Such a phenomenon as its inability to enter on normal activity for speech, or to abruptly stop movement when in action for speech, calls for a definite determination of the reasons for such extraordinary abnormality of action. Dr. Bluemel's assumption that stoppage of the speech machine in stuttering is but a simple delay in its movement, is indicative of his lack of understanding of the peripheral symptoms of stuttering.

A general statement of the peripheral activities of stuttering as I, now, (about twenty-eight years after Dr. Bluemel wrote) understand them, is as follows: Each complete act of stuttering consists (A) of a series of activities of the speech mechanism without resulting sound production; and, immediately following, (B) certain activities resulting in sound production. The A series (without sound production) consists of (1) abnormal activities and (2) normal activities; the B series of activities (with sound production) are all carried on in principle normally. The (1) ***abnormal*** activities of the A series (without sound production) consist of complete arrest of the compressive movement of the chest wall, although the chest musculature continues in action; and the complete arrest of the laryngeal movements, including vocal cords, although all muscular action continues, in principle exactly as stated above for the chest musculature.[6]

While the above described ***abnormal*** activities of the chest and the laryngeal musculature, including the musculature for vocal cord action, are proceeding, the (2) articulative and palatal placements are *normally* made, and the musculature which causes them is kept in action. All muscular activity in series A (without sound production) whether of chest or larynx, which are acting ***abnormally***, or of articulation or palate, which are acting in principle ***normally***, are usually in a state not, strictly speaking, of tension, but of over-action. No sound is forthcoming. All of the above constitutes the first and essential act of stuttering.

The second act of stuttering, series B, involves the placement of the vocal cords in position to produce voice for the sound in mind, while the chest wall enters now into normal compressive movement, thus forcing air against and between the vocal cords to produce the sound psychologically called for. Meanwhile the articulative and palatal positions remain normally placed. The sound is thus normally produced. This entire procedure, first series A, of elaborate

[6]The above statement is correct but not complete. It will be elaborated in a future article.

abnormal muscular action for chest and larynx, and, *normal* action for articulation and palatal movement; and, immediately following, series B, consisting essentially of *normal* action for all systems of musculature of the speech mechanism, are repeated one, or more times, to constitute the characteristic repetitive activities of stuttering. The sound appears, then stops, and appears again, according to the above description of the abnormal and normal muscular acts involved. Dr. Bluemel's conception of the act of stuttering as a mere simple delay of vocal action and then the production of the sound in mind, is seriously incorrect.

Conclusion.—Since the author mistakenly selects for consideration only the normal activity of the act of stuttering, that of sound production, and leaves utterly unconsidered the essential act, that of the stoppage of movement of the speech machine, his statement has no relation of any kind to the explanation of stuttering, and, therefore, is utterly valueless as an "argument."

III. Dr. Bluemel: *"If the stammerer's difficulty lay with the articulation of the consonant he would stammer quite as much at the end of a word as he does at the beginning," and stammering on the end consonant, the author continues, does not occur.*

Dr. Bluemel opened his discussion with the purpose of combatting a theory which attributed the act of stammering to a disturbance in the laryngeal mechanism of voice production. Now "difficulty" of "articulation" becomes the theoretic cause of stammering which he is opposing. Although confusing one can do nothing about this change in the author's conception of his opponent's theory. Now he argues that his opponent's "difficulty" of "articulation" theory as to stammering on the consonant does not explain the absence of stammering on the ending consonant of a word. While he makes no effort to explain how the "recoil" theory meets this difficulty, one may assume his meaning to be that, since there is no vowel in the same word succeeding the ending consonant, on which a "recoil" might occur, therefore the ending consonant could not be stammered on.

In order to emphasize the idea that consonants and vowels behave in principle alike in stammering, a subject to come up again later, I will remark, from my own standpoint, that ending vowels, as well as ending consonants, are also unlikely to be stammered on: for example, *family, degree, today, they, blow,* etc. However, the ending *o* in *radio* and *Romeo,* since it constitutes a final complete syllable, would be more likely to cause trouble. In the extremest, rarest, cases of mature stammering, in which practically every sound may at times cause disturbance, neither of the above statements as to absence of stammering on ending consonants and vowels might hold.

Let us turn to a peculiarity of stammering which in this connection comes in Dr. Bluemel's path, but which he overlooks, apparently because he seems to be so intently bent on proving the "recoil"

theory. I refer to the marked susceptibility for stammering to occur in highest degree (1) on the first sound of the first word, beginning on act of talking; in second degree (2) on the first sound of the first word beginning a sentence; (3) on the first sound of some word within the sentence; and (4) on the first sound of a syllable within a word. The syllabic structure of words serves continuously to create consonant-vowel sequences, and also, but comparatively in greatly diminished numbers, vowel-consonant sequences. These sequences occur within sentences very many more times than at the beginning of sentences. Why should "recoil" occur so predominantly on the beginning word of sentences, even though the opportunities are so many times greater in numbers within sentences? Or why should "recoil" occur so predominantly at the beginning of longer words rather than within such words? Dr. Bluemel passes over the subject. I confidently prophesy that when the real etiology of stammering becomes established these questions will readily find their correct answer.

In the process of developing speech one may imagine that the child conceives of attacking syllables of longer words for their acquirement in much the same spirit that he approaches new words of one syllable. Stammering not only typically occurs at the beginning of a word, but when it does occur within the word, the "recoil" theory of itself alone certainly has no bearing on these facts. From the standpoint of the stammerer the syllable seems to possess something of the character of an entity, similar to that of a word. There seems to be a definite relation between the tendency to stammer on the first sound of a sentence and of a word and of a syllable, and without regard to whether that sound be a vowel, or consonant.

Conclusions.—(1) The predominant susceptibility to stammering on words beginning a sentence, on sounds beginning a word, or within the word beginning a syllable, is not accounted for by the "recoil" theory.

(a) The reason why ending consonants (or vowels) are not stammered on is because of their end position within the word, or syllable, and not because of any reason indicated by the "recoil" theory.

IV. Dr. Bluemel: Stammering often occurs on the beginning vowel of a word; "*therefore the difficulty does not lie with the consonant.*" Later,[7] he states, "*still another fact bears witness that the stammerer's difficulty is not one of phonation. The stammerer often vocalizes when stammering or stuttering upon a vowel.*" "*The stammerer may find 'any' as difficult as 'many,' and 'angle' as difficult as 'dangle'.*" *Indeed* there "*may be no consonant in all the word,*" as *I* and *a*, each of which may be stammered on.

The vowel, then, may be stammered on when beginning a word, or when of itself alone it constitutes an entire word. There can be no

[7] *Ibid.*, p. 86.

question as to the truth of this statement. And one might add that the vowel even within a word, especially when it constitutes the beginning of a syllable, or of itself alone constitutes an entire syllable, may also be stammered on. The author's statement is, to repeat, since the ***beginning*** vowel of a word, or the vowel which constitutes an entire word, is stammered on, "therefore the difficulty does not lie with the consonant."

I have tried most earnestly and diligently to understand what this vague statement means. I have even written out in detail the only meaning that seemed possible to attribute to it; but that attempted explanation seems so meaningless that I have decided not to offer it in this essay. However, one might remark, somewhat quizzically, that if a consonant could constitute a complete word, it too, like the vowel, might be stammered on independently.

The author leaves wide open any complete explanation of why the vowel is stammered on. Stammering is a very different conception from that of "recoil." The reason for stammering on the consonant has already been expressed as follows: "his (the stammerer's) futile struggles with the initial consonant are directed solely by his kinaesthetic imagery, but he cannot pass to the vowel because he cannot recall its . . . sound, in short the ***vowel color***." Thus the stammering on the consonant is due not to one but to two factors, first, the absence of auditory imagery for the consonant stammered on, and second, the delay of the consonant due to the "recoil" of the succeeding vowel. Reasoning from my own observations, vowel and loud continuous consonant are stammered on under identically similar circumstances, and are equally susceptible to the same manner of stammering. Therefore, the psychologic explanation of stammering on each must be identical. Therefore, the stammering on the vowel must be due, first, to the momentary absence of the auditory imagery of the vowel stammered on, plus, second, the "recoil" of some succeeding sound from the mind. But "recoil" occurs only on a vowel, and practically always there is no succeeding vowel in the same syllable, therefore the second reason for stammering on the vowel is absent.

Conclusion.—(1) The statement of the author at the beginning of this section I have been unable to understand; (2) the author has not accounted for stammering on the vowel, an important factor of the speech disorder in question; (3) if stammering on the vowel is unaccounted for, need we trouble further with the "recoil" theory as an explanation of stammering?

The second "argument" in this section reads: Since the stammerer often "vocalizes" when stammering or stuttering upon a vowel, therefore "the stammerer's difficulty is not one of phonation."

Repeating what I have heretofore stated, typical stammering consists in a complicated stoppage of the action of the peripheral speech machine,[8] which, of course, renders phonation for the period

[8]Page 103.

of stoppage impossible. Such typical stammering is often accompanied by production of the sound stammered on, during the moment of recovery from the stoppage. This is so common as to constitute of itself an incidental typical step following the basal act of stammering, which, then, consists of, first, complete stoppage of the speech mechanism, and, second (with irregularity), momentary production of the sound stammered on. Also, as already stated, the production of the sound stammered on as the sole act of stammering, occurs in certain stammerers who, through self-control, have learned how to prevent at times actual stoppage of the speech machine.

Conclusion.—As heretofore, (in discussion of stuttering) Dr. Bluemel again bases his "argument" on the incidental sound production in the act of stammering, and not on the fundamental act, the stoppage of the speech machine while its musculature is in action, which act can, I believe, have no possible reference to the "recoil" theory.

V. Singing "*differs from speech chiefly in the manner in which the vowel is produced, while there is manifestly no radical change in the consonants.*"

I would define singing as the rhythmic, continuous production of attractive, or beautiful, vocal tones of constantly varying pitch, usually through the employment of spoken words, in accordance with the principles of music. The concentration is on perfection of action of the vocal mechanism affecting especially certain loud continuous consonants and the vowel sounds.

Conclusion.—How singing might operate to prevent the "recoil" of the vowel is certainly not evident to me, but that it would prevent impairment in the action of the vocal mechanism is clear, because the mind is concentrated on the continuous production of tones of fine vocal quality.

VI. Dr. Bluemel: "*If the stammerer's impediment were due solely to his inability to produce voice, then all trace of the impediment should vanish as soon as he begins to whisper, for in whispering no phonation occurs,*" and yet stammering occurs on whispered sounds.

In an important work by John Wyllie, M. D.,[9] may be found a scholarly discussion of the history of the discovery of the production of the whispered voice by the impact of the expired air against the vocal cords, especially adjusted for production for that type of voice. Since the time Wyllie's book was published, which occurred in 1894, eighteen years previous to the appearance of the first treatise on stammering of Dr. Bluemel, which book the latter author quotes on other subjects, the above-mentioned knowledge as to the production of the whispered voice has been confirmed many times. This has been done, (1) by observation of the movements of the vocal cords through the laryngeal mirror during the production of the whispered voice; (2) by observation externally by finger palpation, during the

[9]The Disorders of Speech, 1894, Chapter II, page 33.

production of the whispered voice, of the tip of the thyroid cartilage; and (3) by the study in moving pictures of the larynx in action. These observations indicate unquestionably that the production of the whispered voice is as much an act of phonation as is the production of the loud voice.

Conclusion.—This "argument" is valueless because based on a proven untruth.

Words Controverting the Recoil Theory

The writer has not found in any of Dr. Bluemel's books explanatory reference to many words stammering on which is not covered definitely and completely by Dr. Bluemel's theory, namely, words in which there exists no convenient *immediate* vowel following the consonant stammered on, upon the "recoil" of which the stammering could be based. Examples of such words: *clock* (*k-l-o-k*), or *Christmas* (*K-r-i-s-t*), etc., on which stammering would probably always occur on the *k* sound; or *trip*, *tremble*, etc., on which the stammering, often if not always, occurs on the *t* sound; or *plenty*, *prince*, etc., on which the stammering wou.d usually, if not always, occur on the *p* sound; or, and even more significantly, *spring*, *split*, *strip*, etc., on which, in my observation, the stammering would occur always on the *s* or *t*, but never on the *r* or *l*. Dr. Bluemel should have explained why the stammering does not regularly occur on the consonant which immediately precedes the recoiling vowel, or how the effect in producing stammering of the "recoil" of the vowel is so regularly carried back one or two sounds to the earlier consonant in the words mentioned. One is allowed by the failure to explain this phenomenon to strongly suspect that some causal factor beside the "recoil" of the vowel is required for its explanation.

Especial Susceptibility to Stammering on Beginning Sounds of Words

Let us now examine carefully the order between consonant and vowel in the word, from the standpoint of which sound, vowel or consonant, is stammered on.

Vowel preceding	*Consonant preceding*	*Vowel preceding*	*Consonant preceding*
at	tap	aim (a-m)	may (m-a)
eat (e-t)	tea (t-e)	awl	law
old (o-l)	low (l-o)	off (aw-f)	false (f-aw-l-s)
eel	lee	oak (o-k)	coe (k-o)
or (aw-r)	raw (r-aw)	ore (o-r)	row (r-o)

Vowel preceding	*Consonant preceding*	*Vowel preceding*	*Consonant preceding*
ore (o-r)	roar (r-o-r)	it	tip
Abe (a-b)	bay (b-a)	up	pup
		am	mat
ace (a-s)	say (s-a)	if	fit

One might multiply indefinitely these reversions of order of vowel and consonant beginning words or syllables. To one familiar with the symptoms of stammering it is evident that any especial susceptibility to stammer on particular sounds in the above words, whether vowel or consonant, would be determined practically always by its first position in the word. In view of this regularity and importance of relative position in the word in determining the sound to be stammered on, whether vowel or consonant, how can it possibly be conceived that, when a consonant begins a word (as in the second row above), it is not the primary position of the consonant rather than the "recoil" of the succeeding vowel that superinduces the stammering? Under the circumstances to attribute the stammering on the *o* in *old* to "recoil" of the succeeding *l* would seem quite as reasonable as to attribute stammering on the *l* in *low* to "recoil" of the succeeding *o*.

Distinction between Vowel and Consonant

Does such a definite distinction between the psycho-physiology of vowel and consonant as to constitute a real basis for the "recoil" theory actually exist? Dr. Bluemel assumes the existence of such a distinction, but without discussion. Consonant and vowel are acquired by the child at the same time through the imitation of spoken combinations of sounds, i. e., words. The kinaesthetic and auditory memories for each sound eventually become recognized and associated in the child's mind. This is necessary for their definite acquirement. Whether at this early stage in speech development a clear distinction between consonant and vowel exists is hard to tell. Such a distinction would seem to be a necessary requirement of the conception of "recoil of the vowel." As habit gradually takes the place in large part of the use of auditory and kinaesthetic memories for the production of speech, such distinction between vowel and consonant, as may have existed, certainly tends to become vague.

As to the physiologic distinction between vowels and consonants, one should realize that all consonants, like all vowels, are produced by the impact of the breathed air against the vocal bands, properly approximated for each sound; and the air vibrations so produced are in like manner modified by their passage out a distinctive oral channel for each sound. In the course of the thousands of years in

which the evolution of English speech has been developing a selective process, partly a matter of accident, has served to create the present distinction between the consonant and vowel. And yet the sound elements of the vowels have probably been of importance in determining this selective process. However, distinction between vowel and consonant is not based wholly on distinction of sound, but is based also on a physiologic articulative distinction, as follows: The vowel is easily and naturally capable in the articulative act of associative production with any consonant, either preceding or following itself. This possibility is not universally true of any typical consonant, and is necessary for the employment of the vowel as the basis of syllabic speech structure.

As we now consider those consonants which approach in character nearest the vowel the importance of the distinction between vowel and consonant seems for them to grow dim. The continuous loud oral consonants are *w*, *v*, *th* (loud), *z*, *y*, *zh*, *l*, *r*, and the diphthong *qu* (kw). The *w* is but a simple modification of the vowel *oo* (oozing), and the *y* a modification of the vowel *i* (it). The *r* is either vowel or consonant, depending on its use: in *her*, *earnest*, *whirl*, etc. (as spoken in the Central western United States), it is a vowel; in *run*, *read*, *are*, etc., a consonant. I regret my lack of close knowledge of the speech of other languages, but I have little doubt that many English consonants there become vowels, due to slight modification in their manner of production and their sound, and to the practice of other peoples in their use in the production of words.

Considering the identical manner of acquirement of vowel and consonant in childhood and their doubtful distinction in the child's mind, also their like psycho-physiologic production, and the manner in which certain vowels and consonants tend to merge into one another, whether such clear distinction between them as to encourage the truth of the "recoil of the vowel" theory exists, seems doubtful.

Summary and Conclusions

I. When a human psychophysiologic function becomes disturbed in action, it goes without saying that a clear knowledge of the basal normal function is necessary for a definite appreciation of its disordered activities; also that the etiology of such disordered action must explain the symptoms involved; also that, if such abnormal symptoms are not clearly understood, their etiologic explanation is likely to be mistaken; and, finally, that if an author mistakenly picks out certain symptoms of a disorder for etiological explanation, which symptoms fit into a preconceived theory as to cause, while leaving unconsidered other more fundamental symptoms, he can hardly hope to produce a correct solution of the disorder. It seems to me that Dr. Bluemel has built his thoery as to the cause of the impediment of speech in stammering in a measure on each of these fundamentally important errors. Because of increased knowledge, such errors are now at this later date more readily appreciated.

II. The author fails to realize that speech is produced by a complicated psycho-physiologic mechanism, and that its peripheral inability to produce sound while (wrongly) placed in action for speech must depend on seriously disordered psychophysiologic action. His evident idea that the speech machine during such periods merely stops its activity, constitutes a fundamental misunderstanding of the symptoms of stammering. The *actual* reason for the stoppage of the speech machine in stammering could not, I believe, be in any manner related to the "recoil" theory.

III. The author's assumption, because during an act of stammering sound is produced, that the vocal mechanism is acting normally, fails to realize (1) either that a preceding stoppage of the speech mechanism has taken place, or (2) that, through self-control in individual cases, fundamental stoppage of the speech mechanism has been prevented. He seems to select the superficial symptom, the production of sound, that fits into his theoretical needs, and to ignore the more fundamental symptom, the stoppage of the speech mechanism, which does not.

IV. To the author the basal symptoms of "holding" and "blocking" in the speech mechanism during an act of stammering seem not to exist; to him the speech disturbance is merely hesitating until the "recoil" of the succeeding vowel has passed over, so that the speech can be proceeded with.

V. The failure to stammer on the ending consonant of a word (also on ending vowels that do not constitute entire syllables) seems to be due to a fundamental susceptibility to stammering on the beginning sound of words and syllables, present in all stammering, rather than on the "recoil" theory.

VI. In a vaguely worded statement, stammering on the beginning vowel, and on a vowel which of itself alone constitutes a word, is conceived by the author to confirm the theory of the "recoil of the vowel" as the cause of stammering on the consonant. The meaning of this statement is too vague for understanding.

VII. The author fails to make any attempt that I am aware of to explain stammering excepting on the consonant. Since a "recoil" possibility is not afforded to explain stammering on the vowel, one is left to surmise why vowels are stammered on.

VIII. Stammering of course occurs on whispered as well as loud consonants. Dr. Bluemel, who believed that the larynx was not involved in the production of the whispered sound, considered this fact to be a strong bulwark of support for the "recoil" as against the laryngeal theory of cause for stammering. Since, however, whispered, as well as loud sounds, are in fact produced in psychophysiologic principle exactly alike, by the impact of the outbreathed air against the vocal bands properly placed for each type of sound, this item of supposed support completely falls.

IX. The author fails to mention the highly significant fact that particular susceptibility to stammer on certain words is determined

by their position in the expression of thought: (a) the first word at the beginning of an act of talking; and (b) the first word of a sentence while talking. Also he fails to mention a like susceptibility to stammer on certain sounds of the word: (1) the initial consonant, or vowel of words; or (2) of syllables within the word. That this susceptibility is not related to the "recoil" theory is indicated by the fact that a reversal of position of sounds in words reverses also the especial susceptibility to stammer. For example, "Oak (o-k) is my name"; "Coe (k-o) is my name." In *Oak o* is the susceptible sound, and in *Coe* the *k*, and by no possibility could "recoil" on the *o* in *Coe* offset the importance of position in determining the susceptibility of stammering on the *o* in its reversed position in *Oak*. It is position in sentence, word and syllable, rather than "recoil" that determines susceptibility.

X. In certain words in which two or three beginning consonants precede the first vowel, like *plant* and *strip*, the stammering occurs practically always not on the first but on the second, or third consonant preceding the vowel. This delay in the effect of the "recoil" of the vowel strongly suggests the possibility that some influence other than "recoil of the vowel" determines the sound stammered on.

XI. (1) The identical manner of development of vowel and consonant in childhood, and their doubtful distinction in the child's mind; (2) the similarity of their psychophysiologic production—for only laryngeal and articulative and palatal details differ; (3) the creation of certain consonants from certain vowels as their bases, and, finally, the employment of certain sounds as either vowel, or consonant, all these facts taken together, seem to preclude the existence of such a definite distinction between vowel and consonant as to constitute an adequate basis for the "recoil" theory.

Final Conclusion

Writing some twenty-eight years after Dr. Bluemel began his work on the etiology of stammering, we now find that he failed completely to establish a causal relationship between the fundamental symptoms of the speech disorder of stammering and the "recoil of the vowel," a relationship necessary to establish the truth of the "recoil" theory.

Inferential

But if the "recoil" theory, which does make an attempt, although feeble, to consider the symptoms of disordered speech in stammering, what shall be said of theories which make essentially no accurate attempt at all to explain such symptoms? Such theories have been flooding American literature for a quarter of a century. As a matter of course the etiology of stammering can be solved only if the symptoms of the disorder are explained. And the fundamental symptom of the disorder is the "stopping" and "holding" and "blocking" of the speech machine. When a theorist develops a conception of etiology

based on the existence of a "stammering personality," or on the idea that emotional or super-energetic personalities account for stammering, or one based on some manner of general psychologic disturbance, his theory is necessarily untrue unless it explains the psychophysiologic symptoms of the speech disorder, and such symptoms are so precisely definite that no such theory possesses any important likelihood of affording such explanation. This is especially true when the psychophysiologic symptoms of the speech disorder have not even been the subject of close study, which fact has lately been universal for such theorists. In my judgment, no theory of the class suggested has as yet even approached a psychophysiologic explanation of the disorder of the speech in stammering.

It is to be carefully noted that emotion which constitutes such an important role in the general symptoms of mature stammering, and which constitutes so important a factor of the "stammering personality," and which is so generally conceived of as an important cause of stammering, might possibly be explained not as a basal causal factor, but as the natural *result* of the inability of the stammerer to talk smoothly and continuously. The moment that stammering can be explained as an immediate psychophysiologic disorder of the speech, to which any young child of whatever personality, (either with or without causal emotional disturbance at onset) is susceptible, the causal role of any manner of general psychologic abnormality must be abandoned forthwith.

In the meantime the stammerer waits and suffers. Methods of treatment based on such mistaken conceptions of etiology are necessarily ineffective. In the course of medical history certain diseases have awaited from one to many centuries for their etiologic understanding. If the studies of stammering continue not to be directed even *towards* a solution of the fundamental symptoms of the disorder, an infinity of centuries must be required for its etiological solution. Therefore, in a succeeding essay I propose to return to the manner of approach to the problem abandoned by recent authors, and to base a study of etiology on a close analyzation of the speech itself and its psychophysiologic disturbance of action. It is my belief that through no other manner of approach can we hope to solve the problem.

DYSARTHRIA AND ITS TREATMENT

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Dysarthria includes all disturbances of articulation due to interference in the discharge of nervous impulses. It is caused by injury, inflammation, degeneration, or arrested development of the brain; or by peripheral lesions in those cranial nerves which control articulation. Lesions in the cerebral cortex do not cause dysarthria unless the Betz cells in the subcortex of the motor areas are affected: on the contrary other cortical lesions may cause aphasia, insanity, or mental deficiency.

In dysarthria the patient's mental speech processes remain normal and he expresses himself fluently in writing and understands everything he hears or sees if his syndrome is not complicated with aphasia, insanity, or feeble-mindedness which often accompany dysarthria. With the exception of motor aphasia, idiocy, and imbecility, language is more affected than enunciation in aphasia, insanity, and feeble-mindedness. In idiocy there is usually no language to articulate. In imbecility the limited language relates almost entirely to things: there is no abstract thinking. Even in motor aphasia the expression is usually simplified and fragmentary, words are omitted, and what little the patient has to express is likely to consist of automatic, emotional, or reactive speech.

In pure dysarthria, then, articulation is much affected by paresis or paralysis of the muscles of the lips, jaw, tongue, and palate, whereas language itself is little, if any, affected. In order to simplify a very difficult subject I shall confine this discussion to pure dysarthria and assume that no aphasia, insanity, or mental deficiency is present.

The speech pathologist must be careful not to confuse weakness of a muscle with paralysis of that muscle. If a child has a long series of rather severe illnesses at the time he is acquiring speech, his speech muscles, like those of his arms and legs, become temporarily weakened, and the tip of his tongue may fail to rise toward the teeth ridge on such consonants as *t, d, n, l, r*. By the time the child has recovered normal muscular tonus, speech habits have become set, and he continues to pronounce these sounds as if his tongue tip were either tied or paralyzed. Or a child may favor a sore palate after a severe sore throat or the removal of adenoids and develop a functional speech defect which resembles cleft-palate speech or that caused by paralysis of the soft palate.

Paralysis is likely to affect both sides of the body unequally: weakness affects both sides alike. Muscular tonus is normal after the illness which caused the temporary weakness is over, whereas paralysis is either flaccid, with too little muscular tonus, or spastic, with too much muscular tonus. Reflexes like those of swallowing and coughing remain normal when the child recovers from temporary weakness. Atrophy develops in the muscles affected by flaccid paralysis, and reflexes are absent in these muscles. In spastic paralysis the muscles concerned with articulation seem to be pulling against some great resistance and facial muscles not concerned with articulation unconsciously come to their aid. Although the prognosis is excellent for functional articulatory defects due to muscular weakness, it is rather discouraging in most cases of dysarthria.

The intermittent clonic and tonic spasms of stuttering and other forms of dysphemia must not be confused with the more constant increased muscular tonus in spastic paralysis. Both seem to increase in severity in emotional excitement. Dysarthria is usually accompanied by paralysis of other parts of the body which are seldom affected in psychoneuroses.

The symptoms of dysarthria depend upon the site of the lesion, and the severity of the speech disturbance varies with the size of the lesion. For this reason the Nomenclature Committee of the American Speech Correction Association has classified the dysarthrias under the location of the lesions which cause them rather than under the names of the diseases of which the speech disorders are symptoms.

It is impossible, for example, to define a speech defect which is characteristic of multiple sclerosis, because this disease affects different parts of the brain in different patients, and one patient or another possesses every speech defect classified under dysarthria.

Let us review, now, the function of each part of the brain as it affects speech, and give a characteristic description of the speech defect which is manifest in a lesion in each part of the brain.

Innervation of the voluntary muscles which produce speech, like that of all voluntary motion, originates in the Betz cells located in the deeper layers of the motor areas of the cerebral cortex. The Betz cells concerned with speech are stimulated by engrams stored in Broca's center in the surface of the cortex just above.

If all of the Betz cells concerned with speech are destroyed by an extensive lesion in both hemispheres in this part of the cerebrum, speech is impossible, and anarthria results. Although the patient knows what he wants to say and which muscles need to be moved to say it, he has lost voluntary control over these muscles and is unable to move them, even though they are still capable of involuntary movement in certain reflexes.

If there is a lesion in the Betz cells in one hemisphere of the brain or in the nervous pathway known as the pyramidal tract which connects these with the roots of the cranial and spinal nerves, there usually will be hemiplegia—loss of voluntary motion of the speech

muscles and of the arm and leg muscles of the opposite side. There is also more or less spasticity, due to the loss of inhibitory control of the cerebrum over certain lower parts of the brain which initiate muscular tonus.

The destruction of some but not all of the Betz cells concerned with articulation, or of the pyramidal tract leading from them, causes spasmoarthria, commonly known as spastic speech, with hemiplegia, increased muscle tonus, and contraction not only of the muscles concerned with breathing, vocalization, and articulation, but of all facial muscles. This over-exertion is continuous throughout the sentence; the muscles concerned with articulation seem to be pulling against some great resistance, and facial muscles not concerned in articulation unconsciously come to their aid. Syllables are equal in length and are laboriously enunciated. Contractures are often present in the arms and legs. When, as is often the case, spastic speech is a symptom of birth palsy, the damage has been done, and the paralysis is not likely to become progressive. Speech training is usually beneficial, although speech which can be understood with more or less difficulty is all that can reasonably be expected.

Lesions in the basal ganglia, including the thalamus and corpus striatum, and in the extra pyramidal tract which connects certain parts of these with the roots of the cranial nerves, also cause spastic speech with increased muscular tonus, rigidity, contractures, and hemi-athetosis—alternate shift in tonus from one skeletal muscle group to its antagonistic muscle group. Patients are unable to make quick alternating movements of the lips, tongue, jaw, and many other muscles. Athetosis usually causes uncontrolled and meaningless inflections of the voice.

Lesions in the corpus striatum cause paralysis agitans, commonly known as shaking palsy, in which the face assumes a characteristic expressionless, wooden, mask-like countenance known as Parkinson's mask. The accompanying speech defect, known as bradyarthria, is characterized by extreme deliberation with marked hesitation, and by monotonous and spastic utterance, due to hypertonicity of the muscles. The speech becomes fainter and fainter as the years go by until the patient becomes unable either to speak or to swallow.

Chorearthria, sometimes called choreatic stuttering, is another speech disorder manifest in those lesions in the basal ganglia which cause chorea, commonly called St. Vitus dance. A series of involuntary spasmodic movements breaks up the speech in a manner somewhat like stuttering. The vocal cords may suddenly tighten to raise the pitch, the lung muscles may contract to increase the loudness of the voice, and the muscles concerned with articulation may jerk and interfere with the pronunciation of vowels and consonants. A sentence never varies twice in exactly the same manner when repeated. A jerking of the muscles of the arms, shoulders, and face is also manifest.

Lesions in the cerebellum cause disturbance in the co-ordination of the larger muscular group actions, principally of the trunk and

lower limbs, but also of the arms, head, tongue, etc. The gait becomes a wobbling side-stepping stagger, muscular tonicity is reduced, and dizziness is often present. This is the familiar disorder seen in acute alcoholism where the cerebellum is toxically affected. The accompanying speech disorder, known as ataxiarthria or scanning speech, is characterized by division of words, long pauses between words and syllables, and by monotonous, drawling, cadent tones such as one uses when scanning Latin poetry, or a child uses in spelling out his letters. The enunciation of words is usually correct, although the last syllable of a sentence tends to be explosive. Phonation is more affected than articulation. Speech loses its usual dominance over respiration; hence logical breath grouping of phrases is interrupted, and the intensity of the voice gradually decreases through the sentence to a point of exhaustion. The voice may become nasal. As the disease progresses the speech becomes more and more halting, colorless, and unemotional.

Lesions of the pons or medulla oblongata usually affect the roots of the cranial nerves concerned with speech and voice, causing the same flaccid paralysis with atrophy of the muscles concerned with speech and voice as do lesions in these cranial nerves themselves.

Even reflexes are abolished when cranial nerves are badly injured. Lesions in the motor fibers of the fifth or trifacial nerve cause ipsilateral paralysis of the jaw muscles; lesions in the motor fibers of the seventh or facial nerve cause paralysis of the lips; lesions in the motor fibres of the tenth or pneumogastric nerve cause paralysis of the soft palate, pharynx, and larynx, and seriously impair voice quality; and lesions of the twelfth or hypoglossal nerve cause paralysis of the tongue and marked inarticulateness.

The speech disorder which accompanies lesions in the cranial nerves is known as leipoarthria, often called "bulbar speech" or "hot potato" speech. It is characterized by marked elision of syllables with slurring first of the linguals, then of the labials, as "Methist Epistle" for "Methodist Episcopal." The voice is high-pitched, hoarse, monotonous, and nasal in quality. As the disease progresses the speech becomes more and more indistinct until it becomes an unintelligible mumble.

As dysarthria is very difficult to diagnose, it is needless to remark that the speech pathologist should work under the supervision of a neurologist if he suspects dysarthria.

Fortunately, all forms of dysarthria seem to respond to approximately the same kind of treatment:

First, the patient must be taught to face the fact that he can never be taught to speak as rapidly as normal speakers talk. He must learn to become content to speak slowly. Haste only increases inarticulateness. It takes much longer to move partially paralyzed muscles than normal muscles. If the patient tries to speak too rapidly, he will never quite reach any mouth mold for any phoneme, and his speech will be nothing but a mumble.

Second, the patient must be taught to relax as much as possible as often as possible. Tension slows down speech and makes it less distinct. Muscles which do not need to move in pronouncing a given sound should be kept relaxed, and should not be used to help the muscles which do need to contract.

Nerve energy needed to move the necessary muscles must not be wasted on muscles not concerned in a given voluntary act.

The patient must first learn to relax the coarser muscles. This work should be done by a physiotherapist or an electrotherapist under the direction of the orthopedic surgeon. For it constitutes a violation of the law in most states if a speech pathologist thus infringes on the field of the Medical Doctor. Massage is often helpful.

Before taking up speech training, teach the patient to relax the muscles of his larynx and to hold tones in a low-pitched monotone. The instant his voice breaks or changes in pitch or loudness, he should cease holding that tone, pause, relax, and try again. Dysarthric patients cannot hold tones more than a few seconds. Ask the patient first to learn by immediate retrospection what a natural sigh feels like, so that he can make a voluntary sigh at any time very much like an unconscious sigh. After explaining to him that speech differs from whispering only in gently moving the vocal bands a bit nearer together, ask him to make the sigh just the same way with the same mouth mold, but spoken quietly instead of whispered. Explain to the patient that the diaphragm completed its work when the breath was taken in before speaking, and that the resilience of the digestive organs, which were pushed downward by the diaphragm during inhalation, is sufficient to maintain the necessary outflow of breath for speech without any muscular effort on his part. Teach him to keep the muscles of his throat and pharynx as inactive while prolonging this loud sigh as the walls of a garden hose, for any change in muscular tension will alter the tone.

While the patient is learning the loud sigh, teach him the principle of mouth play—of making faces before a mirror without allowing any muscle to stop moving or to become tense. First teach him to open and shut his jaw through a distance of nearly an inch without holding his jaw open or shut for even the smallest fraction of a second: it must keep in continuous, but slow, relaxed motion until the patient tires. Next teach the patient to purse and spread the corners of his lips, alternately, without keeping them pursed or spread for an instant. Then teach him to keep his tongue moving continuously in his mouth without muscular tension. After the patient has learned to keep his jaw, his lips, and his tongue separately in continuous, relaxed motion, have him keep all three in similar motion simultaneously.

When the patient has learned both the loud sigh and mouth play, ask him to do both simultaneously, paying no attention to any differences in vowel quality which may result: the loud sigh does not represent any conventional vowel, nor mouth play any group of

conventional vowels. The pitch and volume of the voice should be kept constant during the loud sigh mouth play.

After the patient has been taught to keep the loud sigh going in a low-pitched monotone during mouth play, he should be told that this requires much more muscular effort than speech, and requested to see how much more easily he can combine certain pairs of conventional vowels which require motion of the jaw without any lip motion and vice-versa. For example he can say "we owe" or "were" over and over again until his breath becomes exhausted, keeping his jaw still but the corners of his mouth in constant motion; or "eye" or "aw-oo," keeping the corners of his mouth still but the jaw in constant motion.

Then have the patient read short sentences composed of words beginning with "h" and with vowels and containing as few consonants as possible, making these sentences nothing but mouth play, and not trying to articulate distinctly.

The third principle, muscle training, should be taught simultaneously with relaxation. This must be done by a physiotherapist or gymnastic teacher, under the supervision of a neurologist. After the coarser muscles have been trained, the finer muscles concerned with speech and voice should be trained to work as efficiently as possible.

In order that the tongue may move symmetrically, the patient should be taught to touch the last tooth on each side of both upper and lower jaws with the tip of his tongue; if it is more difficult to move the tongue to one side than to the other, he should move it much more frequently to the more difficult side. Ask the patient to lap the roof of his mouth with the tip of his tongue from in back of the upper teeth to the center of the hard palate, keeping the tip of the tongue moving exactly along the middle of the palate. Ask the patient to protrude and retract the tip of his tongue alternately. Have him encircle the lips with the tip of his tongue.

Have the patient open and close his lips without moving his jaw, and alternately whistle and smile without moving his jaw.

If the soft palate is paralyzed, have the patient blow out candles, blow up toy balloons or paper bags, play a wind instrument, whistle, clear his throat, gargle, cough, and do any other exercises which encourage the breath to pass out through the mouth rather than through the nose. It often helps to pinch the nose firmly as the exercise starts and gradually release the nostrils as the exercise progresses, so that the patient will hardly know when his nostrils first become free.

The patient must learn to move one muscle group without allowing any other muscle group to move in sympathy. When a patient lifts the tip of his tongue he should not throw up his head, turn his head to one side, or contract any of his facial muscles. He must concentrate on the muscle which he is to move, and make sure, by watching his face in a mirror, that he is keeping all other muscles relaxed.

After the patient has acquired reasonable control over the finer muscles which are concerned with articulation, he must be taught how to form the mouth molds for those phonemes which he consistently mispronounces. He must take each position slowly and thoughtfully with the least possible effort, keeping all muscles not concerned in the motion relaxed. If paralysis makes it impossible to make a given mouth mold, he must be taught the nearest compensation possible by using a muscle which he can control. For example, if a patient cannot raise the tip of his tongue, he will have to be taught to substitute the blade of his tongue for the tip of his tongue; he can place the tip just under the lower teeth and push the tongue forward until the blade strikes the upper teeth or alveolar ridge as desired. He can thus be taught to make an adequate *t, d, n; s, z; sh, zh; ch, j*; and, with practice, even *l* or *r*. If the lips are paralyzed and the jaw is not, the lower lip may be brought against the upper teeth for *f* or *v* by throwing the lower jaw forward or pulling it backward as necessary; *p* can be made by exploding *f* by suddenly lowering the jaw, *b* by exploding *v*, and *m* by making *v* through the nose.

After voice quality and articulation have been improved as much as possible and the new speech habits have become set, it still remains to correct the scanning—the tendency to make every syllable of equal length, to prolong the least important syllable in a sentence as long as the most important syllable is held, and to pause between words and syllables. Experiments with normal speakers at the Harvard University Psychological Laboratory showed that there is a strong tendency to pause between words when all syllables are made of equal length. To run words smoothly together, therefore, it is necessary only to vary vowel lengths as do normal speakers. It is well known that there are three ways in which an accented word or syllable may be stressed; it may be made louder, in a higher pitch, or held longer than adjacent syllables. Dysarthric patients and stutterers tend to overdo the first two methods of stressing accented syllables, and to make all vowels of equal length. They must be taught to speak in a low-pitched monotone, therefore, with the least possible effort, and to touch unaccented words and syllables just as lightly and quickly as possible. They already make accented syllables too long, hence it is necessary to shorten unstressed vowels rather than to lengthen stressed vowels. Although these patients seem to be able to repeat short sentences read in this manner by their teacher, they find it much more difficult to read aloud or to converse in this manner. They seem, as a class, to become easily discouraged, and to give up practicing prescribed exercises long before they have given them a fair trial.

Summary

Dysarthria is caused by flaccid or spastic paresis or paralysis of the muscles concerned with speech and voice. If the paralysis is caused by some progressive disease of the central nervous system, speech training probably cannot be permanently beneficial, and the

patient should be told so frankly by his neurologist before any speech training is begun.

If dysarthria is caused by injury or by a disease which has run its course, speech may be improved sufficiently by speech training to become intelligible, but can probably never become normal, as injured nerves cannot function normally, and it is difficult for patients to train other nerves and muscles to take over the functions of the injured ones.

Often dysarthric patients are easily upset emotionally, develop feelings of inadequacy, and become discouraged easily. It takes the ingenuity of the speech pathologist to motivate them, to keep them interested, and to get them to co-operate.

The symptoms depend upon the site of the lesion rather than upon the disease which caused the lesion.

The treatment of all forms of dysarthria is quite similar and consists in persuading the patient to speak slowly; in teaching him how to keep relaxed; in training first the coarser muscles, then the finer muscles concerned with articulation; in showing him how to make compensatory mouth molds with muscles over which he has voluntary control, substituting for those over which he has no control; and in teaching him to speak in a low-pitched, sigh-like monotone, making unaccented vowels as lightly and quickly as possible.

Do not forget to attend the Convention at Pennsylvania State College September 2, 3 and 4, 1940, before your school starts— Why not make it a part of your vacation?

THE VOCAL CONSEQUENCES OF VARIOUS SURGICAL PROCEDURES FOR RELIEF OF BILATERAL RECURRENT NERVE PARALYSIS OF THE LARYNX

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The present paper is concerned with the vocal consequences of various surgical procedures which are used for relief of bilateral recurrent nerve paralysis or, as it is frequently termed, bilateral abductor paralysis. There are many causes of this condition but what is said here is confined to those bilateral paralyses which follow thyroidectomy and are traumatic in origin.

These cases present themselves to the speech pathologist much less frequently than do cases of unilateral paralysis and there is much less that the speech pathologist can do with them if, indeed, he is in a position to do anything. They are usually seen at one of two stages:

1. When dysphonia or aphonia develop following thyroidectomy. In these cases, two questions arise: What is the likelihood of recovery of voice; and, is there any type of vocal treatment that would hasten recovery?
2. When surgery is under consideration for relief of increasing dyspnea. The question presented here is the probable effect on the voice of the procedures proposed by the surgeon.

Before attempting to present the evidence on which the answers to these questions should be based, it would seem advisable to review briefly current opinion as to the innervation of the intrinsic muscles of the larynx. Exner (4) believed that each intrinsic muscle was bilaterally and doubly supplied by both the superior and the recurrent laryngeal nerves. Onodi (18), on the basis of extensive clinical and experimental evidence, contended that all the intrinsic muscles except the interarytenoid and cricothyroid are innervated unilaterally by the recurrent nerve. He held that the interarytenoid, being a midline muscle, is innervated by both right and left recurrent nerves and that the cricothyroid is innervated by the external ramus of the superior laryngeal nerve. Many recent writers have followed Luschka (14), who differs from Onodi only in that he considers the interarytenoid to be innervated by both the recurrent and the superior laryngeal nerves. Recent work by Lemere (10, 11) would seem to establish the correctness of Onodi's position. Lemere con-

tends, on the basis of considerable experimental evidence, that the branches of the internal ramus of the superior laryngeal nerve which appear to innervate the interarytenoid muscle in reality pass on through to supply the underlying mucous membrane. If Lemere is correct then, except in cases of anomalous distribution, the possibility of which must be kept in mind, all of the intrinsic muscles of the larynx except the interarytenoid and the cricothyroid are innervated unilaterally by the recurrent laryngeal, the interarytenoid is innervated bilaterally by the right and left recurrent and the cricothyroid alone derives its motor innervation from the superior laryngeal nerve. In this case, complete bilateral recurrent paralysis would destroy the function of all intrinsic muscles except the cricothyroid.

The effect of paralysis of the recurrent laryngeal nerve on the laryngeal picture has been the subject of long dispute. It is observed clinically that the paralyzed cord may assume either one of two general positions: a position of adduction (the so-called median or mid-line position); or a position of partial abduction (the so-called cadaveric position). In a series of 132 cases of paralysis from various causes New and Childrey (17) report that the cords were found in the midline position in 73 cases and in the cadaveric position in 59 cases. In the 39 cases in this series in which the paralysis followed thyroidectomy the midline position occurred in 27 cases and the cadaveric in 2 cases (excluding 10 cases in which there was recovery of function). Smith, Lambert and Wallace (21) report 117 cases of paralysis from various causes in which the cords assume the midline position in 34 cases and the cadaveric position in 83. Hsu and Liu (13) report a series of 36 cases out of which the cadaveric position was assumed in 25, the midline in 11, cases. Either the cadaveric or the midline positions may occur immediately, and either may be present terminally. Change commonly occurs from the cadaveric to the midline position. Change from the midline to the cadaveric position is rare. New and Childrey found no instance of it among 217 cases. Smith, Lambert and Wallace found only one instance of it among 235 cases.

As Lemere (12) points out, the midline position has commonly been attributed to an isolated paralysis of the abductors. The possibility of isolated abductor paralysis was pointed out by Semon (20). This may occur as a temporary state, a permanent partial paralysis, or as an initial state in a progressive paralysis that eventually becomes complete. If the midline position of the cords is due to isolated abductor paralysis then the shift which commonly occurs from the cadaveric to the midline position would appear to be a reversal of Semon's Law, while the shift from the median to the cadaveric position which is rarely found should be common. Lemere contends that the midline position is not due to abductor paralysis but is, rather, to be accounted for by a complete paralysis in which the adduction is due to the pull exerted by the non-paralyzed cricothyroid. This explanation would make the clinical findings consistent with Semon's Law.

When bilateral paralysis follows the more common course, there is an initial aphonia during which the cords are abducted in the cadaveric position. Gradually the voice is recovered but as the voice returns, due to the change of the cords toward the midline position, the glottis becomes narrower and respiration becomes increasingly more difficult. If the cords come into fairly close adduction, respiratory distress becomes acute and unless surgical relief is provided death from suffocation may result.

There are, as we have said earlier, two stages at which the patient may present himself to the speech pathologist for advice. The first of these is during the initial aphonia, at which time he is anxious to know the possibility of recovery of voice and demands vocal exercise to hasten recovery. Differential diagnosis is, of course, the province of the laryngologist. Absence of movement in the cords can usually be determined without difficulty by mirror examination but only the laryngologist is in a position to determine whether this lack of movement is due to paralysis or to ankylosis of the cricoarytenoid joint. If the diagnosis is bilateral paralysis, the prognosis, in the goiter cases to which this discussion is limited, is unfavorable. Jackson (8) says that in adults return of motility usually occurs, if at all, within four weeks and never occurs after six months. Hoover (6) reports a case of unilateral recovery after twenty months. It appears that the voice may return at any time from twenty hours to two years but return of voice is usually indicative of a passive shift to the midline position rather than recovery of motility.

Is there anything to be accomplished by vocal training during this period? In my opinion, any attempt to do vocal training is almost certain to do no good and very likely to do harm. The inevitable effect would seem to me to be exercise of the unparalyzed cricothyroid with a consequent adduction of the folds that will make more likely their final assumption of a tight midline position with its accompanying discomfort and danger. Vocal exercise can be used with benefit in cases of unilateral paralysis.

If the cords finally shift to a midline position, surgery is inevitable. The question asked of the speech pathologist in these cases is: What is the effect of the various operations in current use?

Usually a low tracheotomy will have been done for relief of respiratory distress. With the tracheotomy tube closed by the finger or, preferably, by a mechanical valve, a voice of good quality can be produced. From the standpoint of the voice condition, no further surgery is indicated. However, many people find wearing the tracheotomy tube so disagreeable that they persist in seeking a different form of relief. Desire to get away from the tracheotomy tube has motivated experimentation with a variety of surgical procedures.

Evaluation of what we might term the surgical merit of these procedures is obviously outside the province of the speech pathologist. We are, however, as in the case of cleft palate surgery, legit-

imately concerned with the vocal consequences of the various procedures that may be used.

These procedures fall into four groups:

1. Nerve anastomosis.

Attempts have been made to anastomose the recurrent nerve with the descendens noni (5), the phrenic (3), and the pneumogastric (1, 2) nerves.

Successful nerve anastomosis with restoration of function of the intrinsic muscles would be an ideal procedure from the vocal standpoint, but the attempts that have been made have been so unsuccessful that this procedure is now seldom tried.

2. Excision of tissue.

Several procedures have been developed that involve the removal of varying amounts of cord tissue. Representative of these are the methods suggested by Hoover (6,) Rethi (19), and Jackson (7).

It is obvious that in the extent to which the removal of tissue succeeds in providing a more adequate airway it also produces an inefficient glottis from the standpoint of tone production. The best that can be expected from this type of operation is to effect a compromise between the need for a wide glottis for the purpose of respiration and the need for a narrow glottis for the purpose of tone production.

3. Lateral displacement of the vocal cords without removal of tissue.

Molinié (15), Moore (16) and others have suggested procedures for lateral displacement of the cords without removal of tissue.

From the vocal standpoint there is the same objection to these procedures as to procedures involving removal of tissue. Mechanical widening of the glottis will inevitably result in the production of poorer tone.

4. Muscle transposition.

There has recently been reported a new technique devised by Dr. Brien King (9) for the relief of respiratory distress in bilateral recurrent paralyses. This technique is based on an entirely different principle. Instead of removing tissue from the air passage, an attempt is made to obtain abduction of the vocal cords by muscle transposition. The muscle used is an extrinsic muscle of the larynx, the omohoid. This muscle is ligated one-half to three-fourths inches below the hyoid bone, shortened to the proper length and attached to the posterior border of the arytenoid cartilage. The omohyoid muscle is fortunately an accessory muscle of inspiration and consequently contracts on the inspiratory phase. This contraction displaces the arytenoid laterally, widening the glottis. The omohyoid thus functions vicariously as an abductor of the vocal cords. On the expiratory phase, relaxation of the omohyoid permits the cords to come together in the fixed midline position.

I have had an opportunity to study the vocal and respiratory results obtained by this operation on two patients. The first, Sister B, was operated on for goiter in 1921 and again in 1933. Following the last operation there was a marked increase in dyspnea and stridor. The vocal cords were fixed in the midline position. She was unable to do any physical work, her voice was fair but respiratory distress was grave. She was operated on in July. The King technique was used unilaterally and by November she had recovered sufficiently to work and engage in moderate physical activity without fatigue or respiraroty distress and her voice has been sufficiently good to enable her to teach school regularly. The second patient whom I have observed, Mrs. S., age 63, was operated on for goiter in 1927, lost her voice immediately afterward with a recovery of voice after several months. Breathing became more difficult until she finally became a semi-invalid, unable to do her housework or to walk upstairs. She lost about 32 pounds. Her cords were fixed in the midline position and the glottis was very narrow. Two months after the operation she had gained 16 pounds in weight, was able to carry on all the normal activities of the home, to walk and travel without difficulty and her voice was quite satisfactory.

Judging from the results of these cases, which were the first two on which Dr. King's technique had been used, it is possible to utilize the omohyoid muscle as a vicarious abductor of the vocal cords. This method will provide an adequate airway at the same time that it preserves the preoperative voice of the patient. From the vocal standpoint, unless satisfactory nerve anastomosis can be accomplished, the King technique would appear to be the only method now available for restoration of the airway without adverse effect on the voice.

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SYLLABLE-TAPPING, A NEW METHOD FOR THE HELP OF STAMMERERS

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Hundreds of methods for the cure of stammerers have been published in the course of years and none has proved to be the ultimately and universally accepted treatment speech-therapists have looked for. No more has the method described hereunder the pretention to be the panacea for stammering. It only wants to be a method for the technical part of the treatment of stammering, which does not aim at giving more than a simple expedient for the relief of the stammerer's speech from the very moment the patient begins to follow a speech-course.

This method does not interfere with the deeper causes of the disorder and with the various theories about this subject or about the different varieties of stammering. Because it only combats the symptoms and does not attack the original root of the speech-defect, its application remains independent of the question whether the cause of stammering has to be found in a constitutional predisposition according to Dr. Szondi, Budapest[1], in an organic weakness of the cerebral function of the striato-pallidum according to Prof. Dr. Seemann, Prague[2], in an educational mistake during the period of speech-development (opinion of Höpfner-Fröschels[3]), in an insufficiency of the word-finding faculty (Pichon and Borel-Maisonny[4]), in a fixation of wrong childish subjections (psycho-analytic theories) or in any other physical or mental defect.

The *first stage* of the method of *syllable-tapping* consists of teaching the pupil to accompany the pronunciation of the successive syllables by noiseless taps of the fingers of one hand. This movement, which shows some analogy with a very elementary exercise for piano-playing, should be performed with the right or the left hand according to the preference and the handedness of the patient (but always

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with the same hand) and I prefer to have it started from the little finger and then continuing towards the thumb and *not* in the opposite direction. The motives that lead to this "one-way-traffic" will be stated below.

Example—

I'll	*try*	*to*	*speak*	*in*	*this*	*man-*	*ner*
little finger	ring-finger	middle-finger	index finger	thumb	little finger	ring-finger	middle-finger

This manipulation has a certain analogy with an old expedient recommended for the help of stammerers, namely beating time with one's hand. Speech-therapists used to consider this expedient a mere trick of little or no value. The method of syllable-tapping described in this study may in a certain degree be considered a scientifical refinement and elaboration of this expedient. Beating time with one's hand for instance is a rather rough movement, which cannot possibly be combined with an exact syllabization, because pressing down the hand at each syllable would hinder the fluency of speech and reduce its quickness too much, and pronouncing a syllable with each separate up and down movement of the hand (thus combining each "tap" of the hand with two syllables) is too tiring and too complicated an action to be practically applied. Consequently beating time with the hand can only serve to accentuate the *stressed* syllables of the sentence and although this may have a certain regulating effect on the speech rhythm, it does not help to overcome the spasms and it does not produce all the various advantages granted by the method of syllable-tapping.

The main difference between beating time with the hand and syllable-tapping can, however, best be characterized by the word "trick" and "method" themselves. It is the *methodical application* and its *final sublimation* to *ideo-motoric impulses* which raise it from the level of a simple expedient to that of a well-weighed part of a therapy.

This methodical application in the first place consists of the disciplinal teaching of the "one-way-traffic," in order to enable the pupil to apply syllable-tapping quite automatically. The "one-way-traffic" has to be preferred to a to-and-fro movement, because the former is a less complicated movement, which for people with rather stiff fingers or with a lack of motoric feeling is easier to perform than the latter. Beginning with the little finger is, I think, also preferable to the opposite direction, as the insertion of the thumb into this movement is—strictly speaking—a more difficult element than the insertion of the little finger. The reader can easily control this by trying to make a quasi-nervous drumming movement with only four fingers. He will notice that the combination of the four longer fingers without the thumb is the easiest one. Thus it is clear that, for some people, it has a confusing effect to begin with the thumb, whilst, when beginning with the little finger, the thumb will automatically follow the movement.

Moreover this disciplinal prescription of the "one-way-traffic" adapts the method of syllable-tapping to be applied in groups or classes. Of course, when teaching this method, the teacher has to take care that the pupil's speech may not be reverted into an unnaturally scanned staccato, but must keep its natural, fluent legato character. It is precisely this syllable-tapping with the fingers which is a most suitable method for regulating the speech rhythm and for keeping a legato manner of speaking at the same time. Persons whose fingers are too stiff have to make little exercises at the beginning in order to render them more supple. The pupil has to be accustomed not to look at his fingers; the teacher, however, has to take care that syllable-tapping does not fall back into a mere unrhythmical thumbing of the table without connection with the syllabic division of speech.

If the pupil's speech is interrupted by a clonus (involuntary repetition of an initial sound), he has to stop the finger movement and to lower the whole hand and forearm a little, whilst stretching down the fingers on the table. Then, keeping the top of the concerned finger on the table, the finger and the hand are slowly lifted and the "difficult" sound is at the same time calmly pronounced, after which syllable-tapping is to be continued. Because syllable-tapping has a quieting effect, such cloni will be more seldom than in "untapped" speech, and therefore this method of "*sound-lifting*," as I call it, first has a prophylactic and thence a curing effect too.

Apart from involuntary repetition of initial sounds, the stammerer's speech may be interrupted by spasms (toni). For this case the pupil has to be taught to "*divert the spasm*" by stretching down the finger and the whole hand and forearm, in the same way as for "lifting the sound," but with this important difference that now arm and hand should not be relaxed, but strained intentionally, as long as the clonus of the speech-muscles continues; the duration of this tonus will, however, be shortened by the mental action of the intentional imitation of the spasm in hand and arm, and with its cessation hand and arm are to be relaxed likewise and the "difficult" sound is "lifted" in the way described above. Contracting this habit of "diversion of the spasm" equally has a relieving effect on the stammerer's speech, and thence again a prophylactic and therefore a curing influence on the patient.

After these three things: "syllable-tapping," "lifting the difficult sounds" and "diversion of the spasm," have been taught and after the pupil has learned their application to reading, reciting, answering questions, spontaneous speech, etc., he is taught to make the finger movements under the cover of a book, against the palm of the other hand, against the elbow of the other arm, on his knee, on his back, against the wall or the door, in the pocket of his coat, etc. This is the *second stage* of the method, to which, however, the teacher may not proceed, before the first stage is thoroughly mastered, that is to say the pupil, when applying syllable-tapping, should be able to speak fluently in a natural way with only rare cloni and toni, which

he must be able to overcome easily by "lifting the sound" or "diverting the spasm." In this stage the quickness of speech *may* be somewhat reduced, though this is not at all necessary.

The *third stage* is only for those advanced pupils, who, when applying syllable-tapping, may hardly any longer be considered stammerers. It consists in not really making the genuine "tapping" movements, but gradually reducing their force, until the pupil only performs "impulse-movements," which are later on even substituted by mere mental ideo-motoric impulses without any real finger movements. The pupil now only "thinks" or "imagines" the movements, and does not perform them any more. When the stammerer has been able to reach this stage, he can be considered completely cured.

Unfortunately there always remains a still too numerous group of incurable stammerers who must be considered inaccessible to any method aiming at a complete cure of the disorder. These stammerers cannot reach the third stage of the method, but even in these cases syllable-tapping remains an important help, because it enables the stammerer to express himself, though not without any inner inhibition and perhaps not without some disturbances in the form of initial repetitions or spasms, yet with a sufficient facility and fluency to become a sociable person again.

A summary of the principal advantages of the method of syllable-tapping may close this study, the reading of which will, as I hope, induce some American speech-therapists to make a try of this method with their pupils, and to correspond with me about their results. The success of the treatment may then be promoted by:

(1) Its application in groups and classes;

(2) The application of this method by children-pupils in their reading, geography, and all other lessons, when questions have to be answered, etc. Of course the parents and the teacher, if he is not also the speech-therapist, have to be given some information about syllable-tapping and must be told to have some patience with the pupil.

(3) Syllable-tapping can be learned by every non-imbecile adult or child above the age of about eight years, for the pupil must have an idea of what is meant by "syllable." Apart from this condition, it is, owing to its simplicity, suitable for everyone, intelligent or not, intellectually developed or not, etc.

(4) Patients like this method, because they can apply it invisibly to their auditors, it gives relief from the first lesson on in difficult situations and they can keep their normal kind of speech, which does not assume any specially striking accent. Meanwhile the stammerer is satisfied in his natural want of auxiliary movements.

(5) In a number of cases the mere application of the method of syllable-tapping will prove to be sufficient. Even if on the basis of the speech disorder a secondary feeling of inferiority has arisen, this may disappear automatically, as soon as the patient begins to feel an improvement of his speech. In other, more complicated, cases,

syllable-tapping has to be combined with supplementary treatments, such as administering medicaments[5], relaxation[6], general rhythmical cures[7], persuasion and suggestion[8], psycho-analysis, or whatever other important modern method is judged the right one for each special case.

(6) Because from the very first lesson on already the stammerer can be told that in any eventual difficult situation he may try to apply syllable-tapping in order to overcome his difficulties, teaching this method has the effect of giving the patient an *amulet* against his disorder. The teacher need not be afraid of exploiting the suggestibility of the patient for this purpose. The stammerer's *anxiety* with respect to his own speech and to the occasions he has to speak in public or to important persons for instance will diminish by this quasi-magic *amulet effect* of syllable-tapping.

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1. STUDIEN ÜBER DIE ARTIKULATION DER JAPANISCHEN SPRACHLAUTE

(11) Mitteilung:

Einige Untersuchungen über den Einfluss der Dicke des künstlichen Gaumens und der Lingualfläche der Zähne auf die Artikulation.[1]

Ob die Dickenzunahme des Gaumens beim Gebrauch des künstlichen Gaumens (0.03–1.35 mm dick) und die der Lingualfläche der Vorderzähne (0.20–1.34mm) auf die Artikulation der Sprachlaute irgendwelche Einflüsse hat, wurde in dreifacher Hinsicht untersucht, nämlich 1, ob des Palato-und Linguagramm eine Veränderung der Artikulationstelle aufweist, 2, ob subjektiv irgend eine Artikulationsstörung wahr genommen wird, und 3, ob eine etwaige vernehmbare Lautveränderung entsteht. Es ergab sich folgendes . . .

"Kokubyo-Gakkai-Zasshi"
Eine Vierteljahrsschrift der japanischen.
Gesellschaft für Stomatologie. Band XI, Heft 3.

[1]Aus dem IIte physiologischen Institut der Kaiserlichen Tohoku-Universität zu Sendai (Vorstand: Prof. Dr. med. Fujita).

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A STUDY OF TONGUE PRESSURES OF SPEECH DEFECTIVE AND NORMAL SPEAKING INDIVIDUALS[1]

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Previous Experiments

In 1889 Féré tried to determine the strength of the tongue by means of a glossodynamometer, an alteration of a sphygmometer.[2] Rousselot used a similar instrument with weights on a lever, which operated something like a pair of scissors.[3] I. Fellbrich undertook similar measurements with a lip ergograph.[4]

In 1937 Paul Neumayer published the results of his study using a new instrument for measuring the strength of the tongue. He took a mercury manometer, attached to it a thick walled india-rubber ball about the size of a walnut. Readings were secured by introducing the ball into the mouth of the subject, closing the teeth of the subject to isolate the chewing muscles, and pushing up on the ball with the tongue.[5]

Rousselot mentions nothing concerning his results. Neumayer criticizes the work of Féré and Fellbrich on the grounds that their apparatus contained no way in which other muscles, such as chewing muscles, could be effectively isolated and thus kept from figuring in the results.[6]

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[1]A portion of this study was presented by the junior author to the graduate faculty of the Municipal University of Wichita in partial requirement for the degree of master of arts.

[2]Charles Féré. "Note sur l'exploration des mouvements de la lange," *Comptes rendus hebd. des Séances et Mémoires de la Société de Biologie*, Series IX, Vol. 1, 1889, pp. 278–80. (Neumayer's reference.)

[3]P. J. Rousselot. *Principes de phonètique experimentale*, Part 1, p. 160. Paris: A. Welter, 1897–1908. (Neumayer's reference.)

[4]I Fellbrich. "Untersuchungen uber die Zungenkraft ünd ihre Beizehungen zur Zahnheilkunde und Phonetik," Dissertation, Hamburg, 1936. (Neumayer's reference.)

[5]Paul Neumayer. "Die Kraft der Zunge," *Archiv. fur Sprach- und Stimmheilkunde und angewandte Phonetik*, I, Part 3, (July, 1937), pp. 172–82.

[6]*Ibid.*, pp. 172–73.

Neumayer found that the tongue pressures for normal healthy individuals between the ages of seventeen and sixty years ranged about the same, with the readings for males slightly higher than females. In old people of both sexes who clearly showed signs of advancing age the reading was considerably lower. He also found that individuals possessing lowered physiological conditions had considerably lower tongue pressures than normal healthy individuals of the same age and sex. This proved true in all lowered physiological conditions among Neumayer's subjects, as ascertained by a physician; and also proved true in those suffering from lung, throat, and stomach troubles. The same type of reading was shown in war veterans who were in ill health or weak following war injuries.[7]

Concerning these differences Neumayer states that the development of the tongue muscles, like other musculature, is just as dependent upon the physiological state of the individual as it is upon the activity of the muscle group.[8]

Present Experiment

Neumayer states that his apparatus: (a) gives the tongue the opportunity to develop its full power; (b) isolates all other muscles (Buccinator et al) during the measurement; (c) exhibits an unchanging surface upon which the tongue can exert its force; (d) and the exerting force is quickly readable.[9]

A glossomanometer similar to that used by Neumayer was constructed. A mercury manometer was used in which the glass tubes had an internal diameter of 4 mm. and were 14¾ inches high. This manometer was mounted on an upright wooden frame 12 inches high. On the left end of the manometer was fastened an india-rubber tube 3½ inches long. A clamp was attached to this rubber tube. On the right end of the manometer was fastened another piece of india-rubber tubing 3½ inches long. This in turn was fastened to a piece of glass tubing 4 mm. internal diameter and 3½ inches long. On the end of this glass tubing was attached a thick-walled hollow india rubber ball 1¼ inches in diameter. All connections were glued air-tight. A light spring was placed over the rubber tubing on the right end of the manometer. This spring permitted the rubber ball to move freely in all directions without pinching the rubber tube. A common twelve-inch school ruler was mounted between the upright columns of the manometer with its metric scale on the left or reading side.

The left end of the manometer was closed air tight by means of the clamp on the rubber tubing, since it was found in a preliminary investigation that this was necessary in order to get a steady readable rise of mercury with many speech defective individuals.

The tests were conducted in a room which contained only the

[7]Paul Neumayer, *op. cit.*, pp. 176–81.

[8]*Ibid.*, p. 179.

[9]*Ibid.*, p. 175.

operator and the subject to be tested. All individuals understood that no speech was to be required during the experiment.

Sterilized cellophane bags were placed over the rubber ball so that each individual contacted only the sterile surface during the test.

The subject was shown the glossomanometer and told: "See, when I press in on the rubber ball how the mercury goes up. I want you to put this rubber ball in your mouth and press on it with your tongue and see how far you can get the mercury up. I am going to put a sterilized bag over the ball so that you will touch nothing but what has been sterilized. We will do it three times. Do you understand?"

The rubber ball was then placed in the subject's mouth so that its lower surface rested approximately on the middle third of the surface of the tongue. The subject was then allowed to bite down gently with the teeth on the glass tube to isolate the chewing muscles. The position of the mercury was then noted and written down on a card. The subject was requested to hold his breath (closure of glottis) and press against the ball. The reading was taken at the highest point to which the mercury ascended steadily, subtracting the position of the mercury at the start. Three readings were taken in succession.

128 speech defective individuals were tested on the glossomanometer. Of this group 34 did not succeed in raising the mercury, or had such low readings that the results were thrown out.

Statistical information was obtained on 94 speech defective individuals having a total of 281 glossomanometer readings.

This group was matched age for age and sex for sex with a control group of normal speaking individuals. The control group was composed of 125 individuals with a total of 375 glossomanometer readings.

The age range of these groups was from six to forty-six years.

Results[10]

TABLE I

RESULTS OF GLOSSOMANOMETER READINGS—BY GROUPS

	Number of Readings	Average Age	Average Total Readings	S. D. of Total Readings
All Speech Defectives	281	14.7	2.8	1.1
Controls	375	13.8	3.7	1.0
Articulation	132	12.0	2.7	1.1
Controls	132	12.0	3.4	0.8
Stutterers	81	18.3	3.3	1.3
Controls	81	18.3	3.9	1.1

There was a significant difference in the tongue strength of speech defective individuals and their controls. The average reading for

[10]All formulae used are from *Statistics*, by W. V. Lovitt and H. F. Holtzclaw, Prentice-Hall, New York, 1931.

the speech defective group was 2.8, for the controls, 3.7. There was little difference in either group between the respective readings in each of the three trials.

TABLE II

RESULTS OF GLOSSOMANOMETER READINGS—BY TRIALS

	First Reading		Second Reading		Third Reading	
	Av.	S. D.	Av.	S. D.	Av.	S. D.
All Speech Defectives...	2.7	0.8	2.8	1.2	2.8	1.1
Controls.....	3.6	0.9	3.7	1.0	3.8	1.0
Articulation.....	2.7	1.2	2.7	1.0	2.7	1.2
Controls.....	3.2	1.0	3.4	0.8	3.5	0.2
Stutterers.....	3.1	1.1	3.4	1.4	3.5	1.3
Controls.....	3.8	0.8	3.9	1.4	4.0	1.2

A group of 44 individuals from the speech defective group were segregated. These individuals included all the subjects who exhibited articulation, baby-talk, lisping, lalling, or delayed speech among the speech defective group except those with known organic defects. This group was called the articulation group.

TABLE III

SIGNIFICANCE OF DIFFERENCES—BY GROUPS

	Mean	Difference	σ of Difference	Difference / σ of Diff.
All Speech Defectives.....	2.8			
Controls.....	3.7	0.9	0.20	4.50
Articulation.....	2.7			
Controls.....	3.4	0.7	0.21	3.33
Stutterers.....	3.3			
Controls.....	3.9	0.6	0.33	1.82

The articulation group ranged in age from 6 to 36 years and had a total of 132 glossomanometer readings. This group was matched age for age and sex for sex with 44 normal speaking individuals having a total of 132 glossomanometer readings.

27 stuttering individuals with a total of 81 glossomanometer readings were also separated and matched with controls. The age range of this group was from 6 to 36 years.

TABLE IV

RESULTS OF GLOSSOMANOMETER READINGS—BY SEXES

	Number of Readings	Average Total Readings	S. D. of Total Readings
♂ Speech Defectives.....	189	2.7	1.4
♀ Speech Defectives.....	93	2.8	1.1
♂ Controls.....	210	3.8	1.1
♀ Controls.....	145	3.6	0.6

The differences in tongue strength between the stutterers and their controls was not significant. This result may be due to the low number of readings available for tabulation in this group. Further study is indicated.

Sex Differences

No significant differences in tongue strengths between speech defective males and females, or between normal speaking males and females were found. The readings for male controls were slightly higher than for female controls.

Significant differences in tongue strengths were found between male speech defectives and male controls; female speech defectives and female controls; male speech defectives and female controls; and female speech defectives and male controls.

TABLE V

SIGNIFICANCE OF DIFFERENCES—BY SEXES

	Mean	Difference	σ of Difference	Difference / σ of Diff.
♂ Speech Defectives	2.7			
♀ Speech Defectives	2.8	0.1	0.15	0.67
♂ Controls	3.8			
♀ Controls	3.6	0.2	0.08	2.50
♂ Speech Defectives	2.7			
♂ Controls	3.8	1.1	0.13	8.46
♀ Speech Defectives	2.8			
♀ Controls	3.6	0.8	0.11	7.27
♂ Speech Defectives	2.7			
♀ Controls	3.6	0.9	0.10	9.00
♀ Speech Defectives	2.8			
♂ Controls	3.8	1.0	0.14	7.14

The Group Who Failed

34 speech defective individuals did not succeed in raising the mercury or had readings in which there seemed to be athetotic like movements of the tongue, resulting in fluctuations of the mercury with such rapidity that a reading was impossible. There were no failures in the control group.

None of these results were tabulated or used in the investigation, but a discussion of this group is interesting.

12 of these 34 subjects refused or the rubber bulb was so large that it could not enter the mouth.

Investigating the medical and speech histories of the remainder of this group we find:

9 were suspected mentally deficient, including 3 mongols, and 6 subjects in which physicians reported cerebral defects or injuries.

3 were adult aphasics, and 1 an adult who had lost speech following childbirth.

1 was an adult deaf-mute of good intelligence.

5 were severe spastic paralysis cases with good intelligence.

1 was a case of muscular dystrophy.

2 were severe articulation defects in which all consonants, all vowels, and melody, rhythm, and accent were defective.

1 was a stutterer.

Each of this group had at least 12 trials on the glossomanometer with the same results.

Every effort possible was made to make certain that this group understood what was wanted in the experiment. In the speech defective group who experienced no difficulty with the readings were 4 mentally deficient subjects, 1 speechless child with a cerebral defect, 1 deaf-mute, and 7 cases of spastic paralysis.

Conclusions

The question immediately arises: In cases of articulatory defects with low tongue pressures, is the low tongue pressure the result of the speech defect, or is the speech defect partially mediated through a low muscular strength of the elevators of the tongue? It is, of course, clear that most consonantal motions, with the possible exception of those made by the lips, involve an upward movement of the tongue. Thus the measurement of the strength of this upward movement is closely related to speech, for precision and speed in motor movement have been shown to be based upon a fundamental tonicity of the efferent systems.

A positive answer is gained by a study of the case histories of the individuals with articulatory defects. Malnutrition, febrile and wasting diseases are especially prominent in this group. A first possibility suggests itself that such individuals have a low muscular strength of the tongue due to these conditions. This answer is not completely satisfactory however, for with improved physical condition it would logically be expected that the tongue would gain in strength with the gain in health. Thus an individual who has recovered from such conditions should have a normal tongue strength. Neumayer[11] showed that in all lowered physiological conditions there was a diminution in the strength of the tongue. An examination of the articulatory cases presents some evidence that these cases were in such conditions when the examination was made.

A second explanation suggests itself: It is known that strength improves with use of muscular tissue, no increase occurring in the number of fibers, but a change occurring in the size of the fibers. Thus it could be argued that articulatory cases have a low muscular strength of the tongue due to the fact that they do not pronounce

[11]Paul Neumayer, *op. cit.*, pp. 176–81.

certain sounds, and therefore, the tongue is not exercised. The argument, though tempting, is fallacious on the following grounds: 1. Speech is not the most constant activity of the tongue. The most constant is the ever-recurring swallowing movement resulting from the physiological secretion of saliva. It is interesting to note in passing that many of these children drool. 2. Speech being in the nature of a conditioned reflex cannot possibly operate on the basis of a poor structural organization. In other words, the structure must be deficient priori in order for an articulation defect to develop. (neural, auditory, etc.). 3. From clinical records it is seen that the correction of the speech defect proceeds with the utmost difficulty when some provision outside of the speech process is not made for the development of speed and precision based on tonicity. If the strength or lack of it is the result of defective speech, then the strength should rapidly build up with the acquisition of normal or near-normal speech movements. This does not appear to be the case.

On the basis of the above interpretation the following conclusions appear tenable:

1. Speech defective individuals have, in general, a low muscular strength of the tongue.

2. The responsibility for this deviation from the normal controls is found to lie mainly in the articulatory group, although there is some indication that some stutterers and other types of speech defectives also have low tongue strengths.

3. Muscular strength of the tongue is not a sex-linked phenomenon in either normals or speech defectives, in contra-distinction to speech defects which have been found to be sex-linked.[12, 13, 14, 15, 16, 17, 18, 19] Therefore, the role of muscular strength of the tongue in producing speech defects is mediative, not causative.

4. Since speed and precision of motor movement are dependent upon efferent tonicities, for the most successful work in correction of these defects, a direct attempt must be made to improve the muscular strength of the tongue.

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[12]J. M. Fletcher. *The Problem of Stuttering.* Longmans, Green and Company, 1928. pp. 57–8.

[13]C. M. Louttit and E. C. Halls. "Survey of Speech Defects Among Public School Children of Indiana," *Journal of Speech Disorders*, September, 1936.

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[18]Martin F. Palmer and A. M. Gillett. "Sex Differences in the Cardiac Rhythms of Stutterers," *Journal of Speech Disorders*, March, 1938.

[19]Martin F. Palmer and Anna Mae Gillett. "Respiratory Cardiac Arrhythmia in Stuttering," *Journal of Speech Disorders*, June, 1939.

5. The structural implications of the data and interpretations above throw a serious challenge at the theory of the causation of all articulatory defects by mental or emotional abnormalities.

6. Further study is needed, since there were many trends in the data which might become significant if more cases and controls were to be used.

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FREQUENCY AND EFFECT OF HEARING LOSSES IN CLEFT PALATE CASES[1]

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The explanation of the excessive articulative defects of speech in cleft palate children has had its major emphasis upon the degree of anatomical deformity, particularly of the palato-pharyngeal valve. Perhaps justifiably so. Yet, in the re-education of these cases it has been the experience of speech pathologists to note wide variations in the articulative and phonative speech results post-operatively. There are many cases with post-operative palates too short to effect an efficient closure of the post-pharyngeal space yet showing little loss in the articulative function and little nasal resonance. On the other hand, other cases have adequately long and functionally quite competent palates with poor oral tone and marked articulative distortion. Mothers frequently comment that the speech and hearing responses of these children are definitely influenced by the state of the weather—on dry, sunny days responses are prompt and alert and on damp, foggy days the responses are dull and apathetic, suggesting that at times these children are hard of hearing. Oral-surgeons and otologists recognize that cleft palate children are more liable to ear infections than normal children presumably owing to the irregularities in the Eustachian tube or from its increased exposure to infections.

A survey of the literature over the past ten years fails to reveal even one scientific investigation concerning loss of hearing specifically in Cleft Palate cases. (Probably such studies have been made). Hence the following problem was undertaken in the hope of discovering the bearing of the structural anomaly on the auditory factor. The problem of this study was the investigation of hearing loss in cleft palate children compared to the hearing loss of a control group of functional articulatory speech defectives having no anomalies.

The study was designed to answer the following questions:

1. Do cleft palate cases show marked loss of hearing?

2. Is this loss of hearing greater in cleft palate cases than in the control group of functional articulatory speech defectives?

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[1]Presented at the December, 1939, meeting of the American Speech Correction Society, Chicago, Ill.

3. Are there differences between the experimental group and the control group as to hearing losses for
   a. hearing for telephone use?
   b. the understanding of speech?
   c. perception of vowels and consonants?
   d. the learning of speech?
   e. the development of artistic speech?

4. In a comparison of the medical histories of the two respective groups do the cleft palate cases show greater incidence of ear infections than the control group?

5. Is the structural anomaly as important a factor in causing loss of hearing as is generally presumed?

TABLE I-A

FREQUENCY DISTRIBUTION OF HEARING LOSS IN DECIBLES FOR RIGHT EAR
N=44 Cleft Palate Cases; 44 Control Cases.

AMOUNT OF HEARING LOSS	CYCLES PER SECOND															
	64		128		256		512		1024		2048		4096		8192	
	C. P.	Control	C. P.	Control	C. P.	Control	C. P.	Control	C. P.	Control	C. P.	Control	C. P.	Control	C. P.	Control
100		1		1		1		1			1		1			1
90-99																1
80-89										1					1	
70-79											1		1			
60-69											1					
50-59			1							1		1	2	1	1	
40-49				1	3	1	10	2	8	1	3	1	4	2	5	
30-39	5	1	10	1	11	1	9	2	5	2	3	1	8	2	7	4
20-29	5		15	9	14	11	17	21	10	6	16	8	14	6	14	7
10-19	9	6	10	20	13	27	8	17	12	21	14	19	10	20	8	13
0- 9	11	8	7	11	3	3		1	8	11	5	12	4	11	7	13
—10- —19	1	2	1	1					1	1	1	1	1	1	1	5
Total Number of Cases	31	18	44	44	44	44	44	44	44	44	44	44	44	44	44	44
Median	13.3	8.7	22.6	15.0	24.2	17.0	28.2	21.9	21.0	14.7	21.2	14.6	25.0	15.0	24.2	13.0

Selection of Material.

Children referred to the Speech Clinic after surgical repair work done in the Oral Surgery Department of the Children's Memorial Hospital are, as a rule, below the age of five. Since results in an audiometer test cannot be satisfactorily achieved much below this age the number of cases available for study and chosen at random

from the speech clinic was unfortunately limited to thirty-three cases. Six additional cases were obtained from the speech clinic of the Central Free Dispensary of Rush Medical College and five from my private practice. These forty-four cases, twenty-one males and twenty-three females, ranged from four to thirteen years of age with an average age of eight years and eight months.

TABLE I-B

FREQUENCY DISTRIBUTION OF HEARING LOSS IN DECIBLES FOR LEFT EAR

N=44 Cleft Palate Cases; 44 Control Cases.

AMOUNT OF HEARING LOSS	CYCLES PER SECOND															
	64		128		256		512		1024		2048		4096		8192	
	C. P.	Control	C. P.	Control	C. P.	Control	C. P.	Control	C. P.	Control	C. P.	Control	C. P.	Control	C. P.	Control
100													1		2	1
90–99																
80–89																
70–79											1		1	1		
60–69					1	1	1	2								1
50–59	1		1	1			1		1	2	1	2	1	1		
40–49	2	1			3	1	2	2		1	2		4	1	3	1
30–39	1		2	2	8	2	13	3	4	2	7	3	5	3	9	4
20–29	4		6	6	16	11	18	18	18	5	15	7	12	6	9	4
10–19	11	5	27	27	12	24	8	17	11	21	12	21	15	16	16	12
0– 9	12	11	8	8	4	5	1	1	10	12	4	11	5	16	5	19
—10– —19		1						1		1	2					2
Total Number of Cases	31	18	44	44	44	44	44	44	44	44	44	44	44	44	44	44
Median	12.7	7.2	17.5	15.1	23.7	17.0	27.2	21.6	20.5	14.2	22.6	15.2	21.6	13.7	21.1	10.8

At the time this study was initiated, during 1937, Children's Memorial Hospital had no audiometer so the audiometer tests were done through the generosity of Dr. Maurice Cottle. In an effort to keep the results standard he kindly co-operated in testing the six cases from the Central Free Dispensary and the five cases from my private practice. These tests were given by his well trained assistant under his supervision in his private office. A 2A Western Electric audiometer was used recording the results on the Western Electric audiogram blank. Each subject took the test alone, with the assistant operating the audiometer. She was careful to follow a standard procedure, checking and rechecking to make certain that the patient's light signal was in relation to the duration of the sounding tone.

It is well known that this instrument provides an accurate test of auditory acuity for pure tones over the pitch range most important for speech when used under standard conditions. A sound proof room is regarded as desirable but in these cases this was not possible. Further, a single audiogram cannot be relied upon for an accurate measure of speech perception. It was not possible to make frequent retests in order to obtain an average rating for each case.

Although care was exercised to examine only those children free from upper respiratory infections, it cannot be stated with certainty that they were free from any other temporary pathological conditions of the auditory apparatus. However after the test, each of the thirty-three cases from the Children's Memorial Hospital had returned immediately to the hospital for an examination by the otolaryngologist—whose findings at that time were recorded in an accumulative medical chart.

TABLE II

	Mean Percent of Loss of Hearing for 512-1024-2048 Cycles		Standard Deviation		Standard Error of the Mean		Median Loss for 8 Frequencies	
	Right Ear	Left Ear	Right Ear	Left Ear	Right Ear	Left Ear	Right Ear	Left Ear
Cleft Palate Cases N=44	17.0	16.0	12.3	8.1	1.85	1.22	20.3	18.7
Control Cases. N=44	11.7	11.8	13.0	4.6	1.90	.69	13.6	12.5

The cases for the control group were chosen from the speech clinic of the Children's Memorial Hospital. The number eligible for study was larger, therefore it was possible to obtain a fairly exact matching as to age and sex. Though the two groups were identical in age and sex they exhibited marked difference in anatomical and structural configuration. The forty-four cases of the control group had been referred originally to the hospital for care of some disorder or deviation in health other than cleft palate or speech disturbance. The speech disorder or defect was discovered in the routine medical examination and subsequently the child was referred to the speech clinic. All of these cases had functional articulatory disturbances but no anatomical or structural deformity. The routine of audiometric examination was the same as in the experimental group.

Procedure of Comparison.

The audiogram—a curve showing in sensation units the patient's losses or gains, as compared with the normal, in hearing tones whose frequencies range by octaves from 64 d. v. to 8192 d. v. was analyzed

for frequency distribution of hearing loss in decibles for the right ear, likewise for the left ear, and according to the five methods of the Western Electric formulae given by West, Kennedy and Carr.[2]

The results between the experimental group and the control group are presented graphically in the accompanying tables.

TABLE III

COMPARISON OF EXPERIMENTAL AND CONTROL GROUPS IN HEARING LOSS FOR UNDERSTANDING OF SPEECH

N=44 Experimental Cases; N=44 Control Cases.

Algebraic Mean		Median		σ		ϵ_m	
Experimental	Control	Experimental	Control	Experimental	Control	Experimental	Control
15.30	9.40	15.	9.05	9.57	5.10	1.4	.77

TABLE IV

COMPARISON OF EXPERIMENTAL AND CONTROL GROUPS FOR HEARING LOSS OF VOWELS AND CONSONANTS

N=44 Experimental Cases; N=44 Control Cases.

Algebraic Mean		Median		σ		ϵ_m	
Experimental	Control	Experimental	Control	Experimental	Control	Experimental	Control
16.45	9.0	15.75	8.10	9.70	6.10	1.47	.91

The first analysis presents the frequency distribution of hearing loss for each group at each pitch level as well as the medians. Since only one earpiece was used the following Tables I-A and I-B show the hearing loss in decibles for the right and left ear respectively.

Inspection of Tables I-A and I-B show a difference in hearing loss for each ear between the experimental and control groups.

Table II presents the loss of efficiency of the subject's hearing in using the telephone. The mean per cent of loss of hearing at 512, 1024, 2048 cycles per second for the right and left ear are given separately because of the use of one earpiece. The standard deviation

[2]West, R., Kennedy, L., and Carr, A., "The Rehabilitation of Speech." Harper and Bros., New York, 1937, pp. 395–400.

and standard error of the mean of the experimental and control groups are shown.

The third analysis attempted to reduce to a coefficient which would express in percentage loss the patient's hearing for general understanding of the emotional and intellectual meanings of those to whom he listens. The results are presented in Table III. Algebraic mean, median, standard deviation and standard error of the mean of the experimental and control groups are shown.

TABLE V

COMPARISON OF EXPERIMENTAL AND CONTROL GROUPS IN HEARING LOSS FOR THE LEARNING OF SPEECH

N=44 Experimental Cases; N=44 Control Cases.

Algebraic Mean		Median		σ		ϵ_m	
Experimental	Control	Experimental	Control	Experimental	Control	Experimental	Control
14.70	9.5	14.55	8.85	8.76	5.10	1.3	.76

TABLE VI

COMPARISON OF EXPERIMENTAL AND CONTROL GROUPS IN HEARING LOSS FOR THE DEVELOPMENT OF ARTISTIC SPEECH

N=44 Experimental Cases; N=44 Control Cases.

Algebraic Mean		Median		σ		ϵ_m	
Experimental	Control	Experimental	Control	Experimental	Control	Experimental	Control
15.	9.0	11.80	8.75	9.19	5.05	1.3	.76

The fourth analysis shows what facility the patient has in the interpretation of English as a symbolical language code, in its purely intellectualized and arbitrary meanings. A comparison of results are presented in Table IV.

The fifth analysis estimates the hearing necessary for the ordinary control of the patient's own speech and his learning of new speech sounds. A comparison of results are presented in Table V.

The sixth analysis is an evaluation of the patient's ability to analyze all phases of the speech of others for the purpose of developing artistic speech himself. A comparison of results are presented in Table VI.

Results.

These are preliminary results pending the accumulation of more cases and for that reason the significance of the differences has not been computed statistically. There seem to be differences but it may be possible that these are not statistically significant.

Differences were found between the cleft palate cases and the control group in loss of hearing. The comparison showed consistently greater hearing loss for the cleft palate cases in all the analyses computed.

Not only do cleft palate patients show larger hearing losses than the control group but they also show wider variation.

TABLE VII

SINGLE OCCURRENCES OF EAR, NOSE AND THROAT INFECTIONS IN THE EXPERIMENTAL AND CONTROL GROUPS

N=44 Cleft Palate Cases; 44=Control Cases.

Diagnosis	Ears, Otitis Media, Paracentiesis	Nerve Involvement	Conduction Deafness	Earaches	Acute Nasopharyngitis	Anatomical Deviation of Septum Turbinates	Tonsils (Diseased)	Tonsillectomy, Adenoidectomy	Cervical Adenitis	U. R. I. Frequent Colds	Mastoiditis	Infectious Diseases					
												Measles	Scarlet Fever	Mumps	Whooping Cough	Chicken Pox	Pneumonia
Cleft Palate Cases	27 or 61%	3	2	6	11	3	6	3	2	6	1	2	4	2	1	0	3
Controls	16 or 36%	2	2	2	8	1	18	6	6	7	1	2	2	0	0	2	1

Since the results indicated differences, a study of the accumulative medical records was undertaken in an effort to ascertain how much correlation existed between the medical histories and the loss of hearing.

Since these records are accumulative from the time of a child's first examination in the out-patient department, they represent frequent recurrences of ear disturbances. It was decided to record only once the occurrence of otitis media or a cold. Hence the statistics list only the instances—but not repetitions of the same thing. The results are listed in Table VII.

The comparison of the medical histories of the cleft palate group with the control group is not overly impressive at first glance. Both groups seem to be about equally subject to upper respiratory infections severe enough to warrant visits to the hospital outpatient clinic.

However, closer study reveals differences. The cleft palate cases have a larger proportion of acute inflammations of the middle ear and a much larger proportion of chronic suppurative and non-suppurative otitis media necessitating more frequent myringotomy. Likewise acute nasopharyngitis and a slightly greater occurrence of infectious disease appeared.

On the other hand, the control group showed a greater incidence of diseased and enlarged tonsillar and adenoidal tissue than the experimental group.

A comparison of the medical histories with the audiometric examinations indicated that in spite of both groups being almost equally subjected to upper respiratory infections and ear disturbances, the hearing loss was less in the control group than in the cleft palate group. Although the cleft palate group showed more frequent necessity of paracentesis, it is generally agreed that incision of the drumhead does not make any appreciable differences to the transmission of sound as long as the incudemalleal and incudostapedial articulations are intact.

It might be reasonably conclusive therefore that the structural deformity plays the largest role in causing cleft palate children to be subject to invasions of more acute infections and infectious diseases resulting in their greater loss of hearing.

Summary and Conclusions.

The purpose of this study was to compare cleft palate cases with functional articulatory speech defectives in regard to hearing losses occurring for:

a. telephone use.
b. the understanding of speech.
c. the perception of vowels and consonants.
d. the learning of speech.
e. the development of artistic speech.

Further, the medical histories of the two groups were compared to ascertain the degree to which both groups were subject to upper respiratory infections in an effort to ascertain what effect the anatomical deformity had in causing hearing loss.

The following general conclusions were reached:

1. Cleft palate cases showed loss of hearing.

2. The loss of hearing was greater in cleft palate cases than in the control group of functional articulatory speech defectives.

3. There were marked degrees of hearing loss occurring in both the right and left ears for telephone use.

4. There was a greater difference in hearing loss of the cleft palate group for the understanding of speech than in the control group.

5. A difference occurred in the experimental group as compared to the control group in the hearing loss for perception of vowels and consonants.

6. This was likewise true for the learning of speech,

7. As it was for the development of artistic speech.

8. The results of the comparative study of the medical histories of both groups revealed that cleft palate cases were subject to slightly more disturbances and infections of the upper respiratory tract but resulting in a greater loss of hearing than the control group.

Discussion.

It would seem logical from the results of even this limited study that the assumption of oral surgeons and laryngologists of the greater incidence of hearing loss in cleft palate cases should be accepted. There is not only a definite loss of hearing acuity but also a greater loss in auditory discrimination than in cases other than cleft palate. It is indicative that the anatomical deformity not only makes cleft palate children subject to easier invasion of upper respiratory infections but also makes for more serious and disastrous loss of hearing. That the control group almost equally subject to the same amount of upper respiratory invasion escapes such serious loss of hearing might be explained on the basis of the relationship of the patency of the Eustachian tube to the functioning of the hearing mechanism.

The auditory (Eustachian) tube acts as a ventilating shaft for the middle ear and, as it is opened up during each act of swallowing, it maintains the air pressure in the tympanum at the same pressure as that in the external acoustic meatus and keeps the middle ear patent.

Politzer laid such great stress on this subject that his teachings on inflation and care of the Eustachian tube through the nose and pharynx have become traditional and classical.

The last forty years have only served to provide further evidence that the latent patency of the Eustachian tube is disturbed by anatomical variations of the tube itself or of any structure adjacent to it, or by infections and neoplastic involvement of these structures.

Further, it is generally accepted that impaired function of the Eustachian tube affects the sound conducting apparatus and produces a lessened perception of both high and low tones.

Within the limits of this study the objective results seem to indicate that the impaired function of the Eustachian tube over a long period of time due to anatomical deviation of muscles of the palato-pahryngeal valve as is the case in cleft palate children is the major factor that accounts for a great and frequently permanent loss of hearing. Therefore early otological care is indicated in cleft palate children to prevent and guard against hearing impairment.

CONVENTION INFORMATION

(*See pages 184 and 192 for further information*)

PROGRAM

Sessions will be scheduled on Monday afternoon and evening, Tuesday morning and afternoon, and Wednesday morning. One of these meetings will be held in conjunction with the American Association for Applied Psychology, which will meet in State College on the same dates. The Monday evening program will consist of the President's address. Following the President's address, the members will meet with the Applied Psychologists in an informal smoker. This smoker is definitely being planned as a gathering for the ladies as well as for the men. Light refreshments will be served to those who do not care to smoke. The annual dinner of the ASCA will be held on Tuesday evening.

The Program Committee has voted to continue, with certain modifications, the general form of program management, which was used in the last convention. This was intended chiefly to correct certain objectionable practices which would, over a period of years tend to affect seriously the general quality of our meetings.

The membership is assured that:

1. Meetings will begin when scheduled;
2. Participants will be provided with the *entire* amount of speaking time allotted to them; and
3. Last minute disruptive changes in the program will not be permitted.

The experience of last year convinces the Program Committee that these precautions produced a better convention.

It is equally convinced that the following modifications should be made.

1. There should be fewer papers;
2. Each speaker should be allotted *more speaking time*; and
3. Abstracts must be scrutinized even more carefully for quality.

Considerable criticism has come to the Committee concerning those violations of the rules which it did permit last year. For example, several papers, which had been assigned an especially generous amount of time (at the sacrifice of others) ran over their allotment and disturbed the general sequence.

The quality of these offerings, many insisted, certainly did not justify these infractions.

Several persons did not conform to the requirement that abstracts were to be presented in advance. The Committee could not, therefore, be sure that these papers were correctly placed and of satisfactory quality for inclusion.

A number of contributors also delayed turning in their paper copy until weeks after the convention. These abuses make program conduct extremely difficult.

The Council and membership has voted to make imperative the presentation of *abstracts and complete papers*, for publication purposes, *before the convention*. The Committee will carry out these instructions.

The Council voted to have the next convention take the form of a combined clinical and discussion meeting. "A considerable portion of the program," it directed, "shall be given over to clinics, demonstrations and clinical symposia." The Committee will follow these instructions, also.

There has been considerable agitation, from a variety of quarters, for a more representative and democratic conduct of the affairs of the Association. The Committee feels that a first step in this direction is the provision for business meetings at convenient times. These business meetings will be considered equivalent in importance to any other feature of the convention. Hence, the Committee asks very earnestly that *all members consider it their responsibility to attend these scheduled business meetings*. Only such increased attendance and interest will justify these preparations.

A DEMONSTRATION OF GROUP AUDIOMETER TESTS

BONNIE KISER
University of Wisconsin

You have heard an account of the research leading up to the development of the tests which I am to demonstrate this morning. A general account of their construction has also been presented. I propose to show you these tests and to explain briefly the procedure for administering and scoring them.

First we shall hear the phonograph word test for the hearing of speech. When administering this test to children we draw a diagram of the test blank on the board, tell them exactly what they will hear, and fill in several squares for them. Then they put on the phones and simply listen and watch while the administrator fills in on the first line of the blank on the board the numbers which they are hearing. This procedure accustoms them to hearing with the phones and clarifies the instructions for recording what they hear.

The children are told that a whistle is blown before each number is spoken, and that this whistle is a signal to listen for the next number. If they do not hear the number they are to put an X in the square. The first two lines of the test are used as practice lines and are ignored in scoring. Beginning then with line three the intensity drops about 6 decibels per line. I shall play for you just the last four lines of the test because at the lower levels of intensity you will be able to get a clearer picture of the test at the volumes which are most important in the test. (Test record was played.)

We have scored the test in the following manner. The six lines which are considered in the scoring are weighted. An error on line 8 has a weight of 1, an error on line 7, a weight of 2, on line 6 of 3, etc., up to line three which has a weight of 6. That is, one is not penalized as severely for a mistake on line 8 where it is difficult to hear, as he is for an error on line 3 or 4 where the intensity is greater.

The score is obtained by totaling the weighted scores.

Norms in terms of score below which a retest should be given have not yet been worked out.

Now we come to the group pure tone tests. I have here a record which the Sonotone Company has developed, and the test which we are working on at Wisconsin. I shall play both of them so that you may compare the methods of presenting the tone.

The Sonotone test plays the tones in patterns of one, two or three notes. The subject simply indicates the number of times he heard the tone. (Test record was played.)

In our test, the tones are played at a constant intensity of 15 decibels. Some tones rise in pitch, others fall, and still others remain

unchanged. The bottom half of the blank which you have, is used for this test. The child is told to begin with his pencil in the circle at the left-hand side of the first square. If the tone rises in pitch, he is to draw a line to the top dot in the square; if it falls, he is to draw a line to the bottom dot; if it remains constant, he is to draw a line straight across to the middle dot. The rising and falling tones go through a whole octave range, so that pitch discrimination is not a variable here. The child can tell whether the tone goes up or down if he can hear it at all.

The first line of the test is a practice line. The second line uses the octave from 200 to 400 d. v.; the third line, the octave from 750 to 1500 d. v.; the fourth line, the octave from 2000 to 4000 d. v.

We shall not use the record for this test because, in order to amplify it enough for you to hear the tones without phones, we should probably get surface noises, etc., amplified sufficiently to spoil the tones. Instead, Dr. West will run the oscillator and I shall give the directions in exactly the same way that they are recorded.

The instructions for this test are similar to those for the word test. A sample blank is filled in on the board while the child watches and listens.

We shall omit the practice line this morning and begin with the first line of the actual test. (Demonstration followed.)

The test may be scored by simply totaling the errors and assigning a score on the basis of that total. We have obtained more valid results by demanding that the child get only one of the two presentations of each tone correct. For instance, on line one there are two presentations of a tone which begins at 200 and sweeps up to 400; there are two presentations of a tone beginning at 400 and sweeping down to 200; there are two at 400 constant; and two at 200 constant. If the child gets one out of the two of each type of tone correct, he is given full credit. When scoring by this method, one should probably demand a perfect score.

The first method seems to penalize the child unduly for chance errors—we found too many failing this test who had excellent 2A records. Perhaps our system of recording makes the child a little unsure of what he hears. In the meanwhile, however, while this problem is being worked out, we do know that the second method of scoring yields results much more comparable to the 2A score.

This test has been administered very successfully to third graders; with children younger than that, the results are not quite so reliable, but they are usable.

The work on these tests is far from finished. They should be administered to more children, the recording (especially of the pure tone test) should be improved, perhaps different methods of presenting the sound stimuli should be used. However, it is hoped that the demonstration has served to point up and clarify the theoretical discussion. In any case, you have the report of what we have done at Wisconsin in the field of hearing testing to date.

DYNAMIC vs. STATIC PHONETICS

JAMES L. BARKER, Lic. es Let. Switzerland,
Director of Phonetics Laboratory, University of Utah

In Shaw's *Pygmalion*, a flower-girl is taught to speak like a duchess. In the French Conservatory, provincial Frenchmen acquire a Parisian accent. Phonetics—by this I mean the phonetics of Sweet, Jones, Viëtor, Rousselot, Jespersen, Grammont, and others—has succeeded reasonably well in correcting dialectal pronunciations within a language. Phonetics has even succeeded in teaching the pronunciation of certain foreign languages to other language groups; for instance, it has been successful in teaching French pronunciation to Russians, Italians, Spaniards, etc., but it has been unsuccessful in teaching French or Italian pronunciation to English-speaking people. This fact has been largely overlooked by phoneticians, and the theories of traditional phonetics seem incapable of explaining it. Henceforth, I shall refer to the traditional system of phonetics with which we are all acquainted as "static phonetics."

The basic theories of static phonetics are:

1. Differences in position are responsible for brogue differences.

2. Aside from speed, it is of no consequence how these positions are reached and left, because

3. If the end-positions are corrected, the shifts in position are predetermined and made in the same manner in all languages.

4. The effort to expel the breath within the breath group, or at least within the syllable, is continuous.

In sharp contrast dynamic phonetics asserts that:

1. There is a wide range of positions in which consonants and vowels may be pronounced, and the slight differences in position are effects and not causes of acoustic differences at all.

2. It is of the utmost importance how positions are reached and left in respect to the effort to expel the breath, and these positions are not taken in the same manner in all languages.

3. The effort to expel the breath within the syllable, with the exception of most English syllables, is not continuous and uninterrupted.

4. How positions are taken and left, how long they are held, the rate and direction of movement, and the relative order in which positions are taken are considered as the causes of most brogue differences.

The following quotations are representative of the basic theories of static phonetics:

Passy says: " . . . Many of these (sound) combinations, however, cannot be made without producing intermediate sounds: thus, if we say *ba*, *b* is pronounced with the lips closed and *a* with the mouth wide open; there is therefore necessarily an instant between the two during which the lips are slightly open, i. e., in the position for the production of *v*. If the lips are opened quickly, this *v* is not heard; but if they are opened very slowly, a weak *v* may be noticed, or more accurately, a succession of all the sounds intermediate between *b* and *a*."[1]

On changing position from sound to sound, Jespersen writes: "These on and off-glides have—with few exceptions—very little practical interest. They take place so quickly, that the ear receives no particular impression of them, and indeed (they take place) always in such a manner, that the shortest path between the two positions is chosen, and that they (the on and off-glides) are unfailingly determined, if the end-positions (the positions of the sounds) are given. If two consecutive sounds have a position of the organs (one element) in common, this position is retained from one of them to the other: in *Afva* I do not take the trouble, between *f* and *v* to take the lower lip away from the upper teeth . . . or to cause the breath stream to cease."[2]

"Such a word as *cat* consists not only of the vowel and the two consonants of which it is made up, but also of glides or positions between these sounds. The glide from the initial consonant to the vowel consists of all the intermediate positions through which the tongue passes on its way from the *k*-position to that of *æ*. The number of these positions is infinite; but they are all implied by the mere juxtaposition of the symbols of the fixed sounds, it being assumed that in all transitions from one position to another the shortest way is taken."[3]

Panconcelli-Calzia is of the opinion that "phonetically considered there are no syllables, no words, and no sentences but only more or less lengthy sound groups which are spoken or sung during the one expiration and whose limits lie between the end of one inspiration and the beginning of the next inspiration. . . ."[4]

Others, among them Stetson,[5] limit the continuity of the effort to expel the breath to the duration of the syllable.

Since it is believed according to static phonetics that the path from one sound to the next is determined unfailingly by the end-positions—the positions of the sounds—it has seemed to some phoneticians that these end-positions are alone important and that the

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[1]Paul Passy, *The Sounds of the French Language*, p. 88. Translated from the French by D. L. Savory and D. Jones.

[2]O. Jespersen, *Lehrbuch der Phonetik*, Dritte Auflage, pp. 166–167.

[3]Henry Sweet, *The Sounds of English*, page 53.

[4]Panconcelli-Calzia, *Experimentelle Phonetik*, p. 23.

[5]Stetson, *Motor Phonetics*.

manner of reaching them and leaving them, determined in all languages by the least effort and the shortest path, may be neglected in any corrective treatment of brogue. The older phoneticians ascertained these end-positions by crude measurements, by looking into a mirror, and by the muscular sense. More recently the artificial palate and the X-ray have been used. The theory of correction, however, of static phonetics has remained unchanged. It is indicated by Rousselot in speaking of the use of the artificial palate:

"The *artificial palate* serves whenever the tongue must be applied to the palate. In most cases it is enough to show the contact made by the correct articulation; if not, one makes an artificial palate for the person to whom the articulation must be taught, and one causes him to see wherein the error consists."[6]

This reduces the correction of brogue to the correction of the "end-positions."

Much of the detailed description of English consonants in static phonetics could be applied equally well to the corresponding consonants of other languages. For example, Daniel Jones describes English *p* as follows: "In pronouncing the *p*-phoneme . . . the air passage is completely blocked by closing the lips and raising the soft palate; the air is compressed by pressure from the lungs, and when the lips are opened the air suddenly escapes from the mouth, and in doing so makes an explosive sound; the vocal cords are not made to vibrate."[7] This is as true of German, French, or Italian *p* as it is of English *p*, and consequently of no value in brogue correction. However, Jones does state that "a noticeable puff of breath or 'aspiration' (i. e., a slight *h*) is heard after the explosion of the *p* and before the beginning of the vowel;" that this "puff" is less strong in certain dialects than in others; that it is stronger in the pronunciation of Frenchmen than in English pronunciation. But for these acoustic differences no physiological, mechanical, or other causes are indicated. Moreover, no attempt is made to distinguish either physiologically or acoustically between English and French or Italian *b*. No attempt is made to establish a relationship of cause and effect between physiological means of production and acoustic quality, perhaps, due to the fact that *p* and *b* produced with the lips, have the same position in English, German, French, and Italian. At least, this seems probable, since in the case of *t*, Jones observes that Frenchmen, Italians, . . . use a *t* articulated "by the tip of the tongue against the upper teeth. They pronounce a dental consonant instead of an alveolar consonant. This articulation produces a very unnatural effect when used in English, especially when *t* is final, as in *what wot*."[7]

"Subsidiary members of the English *n*-phoneme exist, and notably an advanced (dental) variety which is used when *θ* or *ð* follows (as in *enthusiasm* [inθju:ziæ zm], *in there* [in ðɛə], and a retracted variety used before *r* (as in enrol [inroul]). Practically

[6]Rousselot, *Phonétique Expérimentale*, vol. II, pp. 1110, 1111.

[7]Jones, *An Outline of English Phonetics*, p. 127.

these varieties are of no importance, because they are acoustically almost indistinguishable from the principle *n* . . ."

"Some foreigners, chiefly speakers of Romance languages, regularly use a dental *n*, i. e., an *n* articulated by the tip of the tongue against the upper teeth. The difference of sound is unimportant except in final position, where the use of dental *n* gives an unnatural effect to English ears."[8]

According to static phonetics the only cause for the difference in acoustic effect between the *n* produced by English speakers and "speakers of Romance languages" is the difference in tongue-palate (or tongue-teeth) contact, and yet the articulation of the sound "by the tip of the tongue against the upper teeth," also encountered in English pronunciation, "gives an unnatural effect to English ears" when used by "speakers of Romance languages . . . in final position." This objectionable *n* of "speakers of Romance languages" can be produced according to dynamic phonetics in any position in which English *n* is pronounced.

Is the place of tongue-palate (teeth) contact responsible for the brogue difference?

According to dynamic phonetics it may be said that there is no one exclusive position for *n* or any other consonant, say *l* for example, in English, French, German, and presumably in other languages. By direct observation it may be seen that the tongue touches farther back for *l* in *pull, milk,* and for the first *l* in *call Albert* than in *play, wealth, health, tell them,* and *tell me.* The *l*'s in the latter list of words are pronounced far forward in what is considered French or even extreme forward Spanish position.

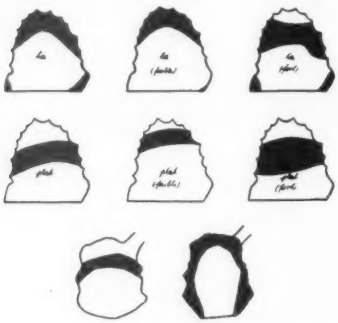

Fig. 1.

La isolated and combined with *p*. The articulation has been produced with three degrees of intensity: medium, weak, strong (Rousselot).

Pull and *play* (Barker).

Rousselot reproduces[9] tracings of the pronunciation of *la* and *plat*. See Figure 1. In one of his cuts of French *l* in *la* (the one at the upper right), *l* is pronounced in what Rousselot gives elsewhere[10]

[8]Jones, *An Outline of English Phonetics*, pp. 155, 156.

[9]Rousselot, *Phonétique Expérimentale*, vol. II, p. 959.

[10]Ibid, p. 916.

as the English position, and all the *l*'s in *plat* are pronounced in this so-called English position, but these *l*'s nevertheless are French *l*'s.

Daniel Jones reproduces palatograms (see Figure 2) for English *l* in which the tongue does not touch back of the teeth as in the Rousselot palatograms, but on the teeth as in the case of Rousselot's

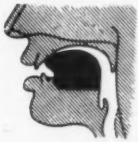

Fig. 79. Tongue-position of 'clear' l (lj).

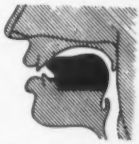

Fig. 80. Tongue-position of 'dark' l (lu).

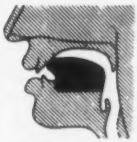

Fig. 81. Tongue-position of intermediate l(lə).

Fig. 82. Palatogram of lⁱ with tip of tongue placed as in English.

Fig. 83. Palatogram of lᵉ with tip of tongue placed as in English.

Fig. 84. Palatogram of lᵉ with tip of tongue placed as in English. The palatogram of lɔ: (l with resonance of English long ɔ:) is very similar to this.

Fig. 85. Palatogram of lɅ with resonance of English short ɔ with tip of tongue placed as in English.

Fig. 86. Palatogram of lu with tip of tongue placed as in English.

Fig. 87. Palatogram of lə with tip of tongue placed as in English.

Fig. 2

palatograms for French *l*, Figure 1. Daniel Jones says further that "variations in the position of the tip of the tongue do not appreciably affect the quality of *l*-sounds. Variations of the quality of *l*-sounds are due chiefly to the position of the main part of the tongue,"[11] but Jones offers no supporting evidence. He distinguishes

[11]Daniel Jones, M. A., *An Outline of English Phonetics*, pp. 159–164.

two main varieties of *l*, clear *l* and dark *l*, and indicates that foreigners substitute clear *l* for dark *l* in English. However, Jones abandons here the static theory of tongue-palate position for *l* held by Rousselot and which he himself maintains for *t*, *d*, and *n*. Dynamic phonetics maintains that certain foreigners do more than substitute clear *l* for dark *l*. They substitute French *l*, Italian *l*, etc., for English *l*, and no variety of English *l*, whether it be clear *l* or dark *l*, whether the back of the tongue is raised or not, is like French *l*, Italian *l*, or Spanish *l*.

Moreover, if Jones and Rousselot can differ so widely as to where the tongue-palate contact for English *l* is made, can one place much reliance in palatograms?

One may say with Dr. Moses of subjects for palatograms that "No two persons make the same tongue-palate contact for the same sound or sound combination, and that no person makes the same tongue-palate contact for the same sound in the same way, even on the same day."[12]

However, if an even more irrefutable demonstration were needed of the inaccuracy of the theory of position of static phonetics, namely, that the position of the tongue-palate contact is responsible for brogue differences which can be corrected by changing the various tongue-palate contacts, it is easily to be had. It is possible to pronounce French *l* in the so-called English position and English *l* in the so-called French position. In fact, one can pronounce either French or English *l* with any tongue-palate contact with which it is possible to pronounce any *l* whatsoever. What is true of *l* is true of *t*, *d*, *n*, and of all other consonants. Consequently, it would seem difficult or impossible to maintain either that the slight difference in position of tongue-palate contact determines the distinctive quality of the sound or that brogue can be corrected by changing the place of tongue-palate contact.

What about the second assumption of static phonetics that the changes in position are of no consequence because they follow the shortest path of least effort?

A glance at comparative kymograms shows that they do not follow the same path. In the English word *crank*,[13] Figure 3, the tongue makes almost simultaneous curves for both *k* and *r*. In the

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[12]Dr. Elbert R. Moses, Jr., Ph. D., "Palatography and Speech Improvement," *Journal of Speech Disorders*, June, 1939, Jr. 114.

[13]In the kymographic experiments reproduced in this article, the lines L (lips, FT (front tongue), and BT (back tongue) record the respective movements of tongue and lips, the lines rising as the movement of closure of lips or tongue compressed bulbs placed on the tongue or between the lips. The lines M (mouth) and N (nose) record air pressure and voice vibrations taken through the nose and mouth, respectively. The line S (spirograph) records the air pressure taken in the mouth back of the point of contact of tongue and lips. At times it also records the vibrations of the larynx.

French word *crâne*, Figure 4, the *k* has a voiceless, then voiced, explosion, and the separate tongue curve for the *r* follows. It is seen at once that the path followed in getting from *k* to *r* is not the same in French and English.

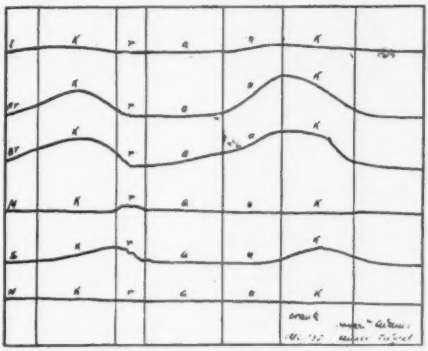

Fig. 3. In *kr* in *crank* it may be seen that the curves are almost simultaneous for the *k* and *r* and that the *k* is exploded directly into the *r*.

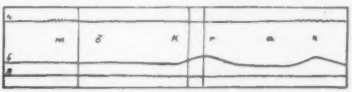

Fig. 4. In *mon crâne* the first part of the explosion of the *k* is voiceless. The vertical lines inclose the voiced part of the explosion. The tongue line T rises for the production of the *r*.

In the kymograms of the pronunciation of *pl*, Figure 5, it will be noticed in the English pronunciation that at the moment the lips are separating to pronounce the *p*, the tongue is nearly in contact against the teeth or gums to pronounce *l*. In the German and French pronunciations, the *p* is exploded some time before the tongue makes contact for the *l*. The transition from the end-positions represented by the lip-contact for *p* and the tongue-palate contact for *l* does not follow any one predetermined path in the three pronunciations.

What happens may be seen directly in a high-speed film of 128 exposures per second. The successive frames have been printed on paper and arranged to read from left to right down. In the English

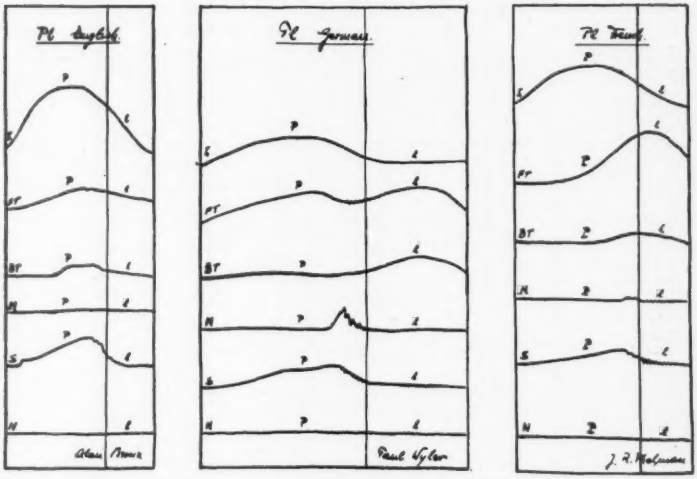

Fig. 5. The vertical lines in these kymograms mark approximately the separation of *p* and *l*. In the kymogram for the English pronunciation of *pl* the lines L and FT rise almost simultaneously. The *l* is prepared with the tongue before the *p* is exploded, the *p* being exploded into the L causing the first part of it to be voiceless. In French, contact for *l* is not completed until some time after the *p* has been exploded. In the German pronunciation of *pl* the lines L and FT indicate a still greater separation of the two consonants than in French. No one "shortest" path is followed in the three pronunciations.

pronunciation of *place* (Figure 6) one may notice that the under-surface of the tongue is seen as soon as the lips open, because the tongue is about to make or has already made the tongue-palate contact for *l*, and the movement of the tongue as it leaves the palate is seen to follow.

Explanation of Figure 6.

The American pronunciation of Miss L. of *pla(ce)* in the sentence: "The place below is to blame." In frames 1 to 11, *the* ðe) is being pronounced and at the same time the lips are closing for the *p* in *place*. In frames 12 to 23 the lips are closed for the *p*. In frame 24 the lips break contact as the *p* is exploded. As soon as the lips separate sufficiently, the tongue is seen to be already in position for *l*. The *p* is exploded into the *l*, the *l* serving as a "vowel" by means of which the *p* is pronounced. In frames 25 to 38 the *l* and the *a* (e) are pronounced successively, but nevertheless the pronunciation is continuous and they are merged together.

FIGURE 6

FIGURE 6

FIGURE 6

In French *pleine* (Figure 7), at the moment the lips separate, the upper surface of the tongue is seen as it rises to make the tongue-palate contact, which it reaches in frame 19. Then follows the downward movement of the tongue as it leaves the point of contact. Again the movement of transition from the point of lip-contact of the *p* to the tongue-palate contact for *l* does not follow the same path.

Explanation of Figure 7.

Plei(ne), pronounced by Mme. M. in the sentence: "Oui, oui, elle boit dans une coupe pleine de bière." From approximately frames 1 to 12 the lips are closed for *p*; in frames 13 to 15 the lips are open for the explosion of the *p*; in frames 16 and 17 the tongue is moving into position for the *l*; in frames 18 to 20 the position of *l* is held; in frames 21 and 22 the explosion of the *l* is shown. In frame 23 the tongue is taking position for the vowel ɛ. In frames 24 to 32 the vowel is being pronounced.

FIGURE 7

FIGURE 7

An examination of numerous kymograms, oscillograms, and films reveals that no two transitions follow exactly the same path. Nevertheless, received pronunciations of English, German, French, etc., do conform rather closely to respective types of transitions; consequently, the manner in which shifts in position from sound to sound are made is highly characteristic of a language; and this would seem to make the theories of static phonetics, namely, that how these positions are reached and left is of little or no consequence, and that they are predetermined if the end-positions are corrected, absolutely untenable.

The fourth assumption of static phonetics remains to be considered: The effort to expel the breath within the breath group or at least within the syllable is continuous and uninterrupted.

Experimental evidence has already been published[14] showing that this theory is true for most English syllables and not true for French syllables. To summarize briefly: English Pronunciation of French is characterized by the production, after nasal vowels and before certain consonants, of nasal consonants not intended by the speaker. For example, in the English pronunciation of French *tombe* an *m* is produced before *b*, not heard in the French pronunciation of the same word. The absence of *m* in French is explicable if the expulsion of the breath is suspended while the shift in position is being made from nasal vowel to consonant. The unconscious production of the nasal consonant is explicable in the English pronunciation only on the assumption that the expulsion of breath from nasal vowel to following consonant is uninterrupted and continuous. Static phonetics has no explanation of this characteristic of the English pronunciation of French.

Evidence has also been published[15] that the effort to expel the breath from consonant to vowel is continuous in English and suspended in French. To summarize again: Kymograms, oscillograms, and direct photographs show gradual merging of consonant and following vowel in the pronunciation of English. In French pronunciation, in the case of an oral consonant followed by a nasal vowel, the explosion of the consonant is seen in the mouth line before the nasal vowel begins; in the case of a nasal consonant followed by an oral vowel, the explosion of the consonant is seen in the nasal line before the oral vowel begins; and in the case of a nasal consonant followed by a nasal vowel, the explosion of the consonant begins at the same time in both mouth and nose lines. All of these peculiarities would be produced if the effort to expel the air is continuous from consonant to vowel in English and interrupted in French. Static phonetics proposes no explanation of these differences.

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[14] *Modern Philology*, "End Consonants and Breath Control in French and English" by James L. Barker, Nov., 1916.

[15] *PMLA*, "Beginning Consonants and Breath Control in French and English," James L. Barker, Dec., 1934.

The dynamic theory of breath control in relation to movement explains a difference in double and cognate consonants that has been overlooked by static phonetics. Grammont says:

"It is not exceptional to encounter double consonants in which the pronunciation produces the impression of a consonant emitted twice in succession. In fact, it is only a prolonged consonant of which *the implosion instead of being immediately followed by the explosion is separated from it by a more or less lengthy stop.* This phenomenon is met with in grammatical forms as:

je courrai, tu mourras,

in words which have lost an unaccented *e*:

il serr(e)ra, nett(e)té, verr(e)rie, enbaum(e)ment,

(and) in the juxtaposition of words (with or without the dropping of an unaccented *e*):

en d(e)dans, il l'a, ell(e) le dit, chaqu(e) quartier, tu gratt(es) ton papier, il pass(e) son temps, un tub(e) brisé, ca ne coup(e) pas, ce n'est pas l(e) lieu, partir résolument. . . .[16]

Daniel Jones agrees with Grammont concerning the nature of double consonants: "Sometimes plosive consonants are not fully pronounced . . . In *that time* (ðæt taim), *red deer* (red diə), the first *t* and *d* are not exploded; in fact the only difference between the *tt*, *dd* here and the *t*, *d* in *satire* (sæ taiə), *red ear* (red iə), *readier* (rediə), is that in the former cases *the stop is very much longer than in the latter.*

"The group *dt* in *bedtime* (bedtaim), only differs from the *t* in *better* (betə, in having a *longer stop*, the first part of which is voiced. In *bedtaim*, *ðættaim*, the stops are of the same length, but in the former the first part of the stop is voiced and the second part voiceless, while in the latter the stop is voiceless throughout."[17]

In both French and English, according to static phonetics as stated by Grammont and Jones, if the stop between the implosion and the explosion of a consonant is very brief, the implosion and the explosion produce the single effect of one consonant on the ear, but if an implosion and an explosion are separated by a long stop, both implosion and explosion produce separate effects on the ear, and what is in reality, according to static phonetics, a long consonant, is heard as a double consonant.

However, if Jones and Grammont err in the statement of fact, the static theory of double consonants itself cannot be true. Observe in the kymograms of *did Tom*, Figure 8, and *Pope Pius*, Figure 9, that double and cognate consonants in English have as short a stop as single consonants.

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[16]Maurice Grammont, *Traité Pratique de prononciation Française*, p. 89.

[17]Daniel Jones, M. A., *An Outline of English Phonetics*, pp. 143, 144.

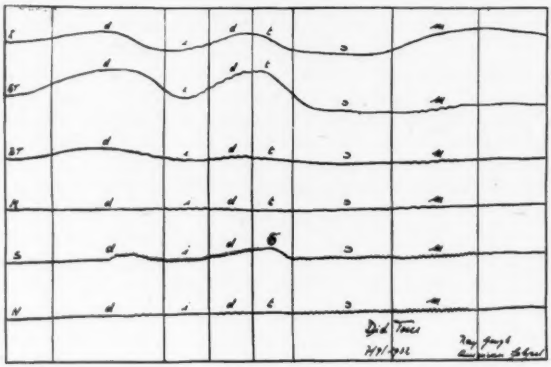

Fig. 8

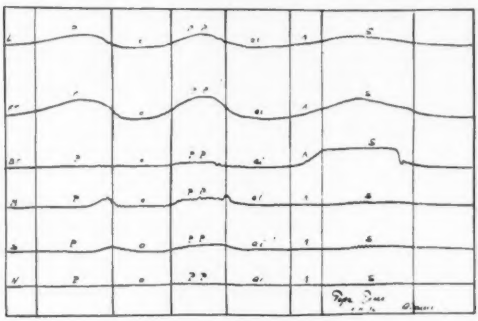

Fig. 9

Figs. 8 and 9. In English *did Tom* and *Pope Pius* the lines FT and L show but one curve for the two consonants *d* and *t* and *p* and *p*. The line FT in Fig. 8 for *dt* is actually shorter than the line for the initial *d* of *did* in the same figure, and the line FT for *pp* in Fig. 9 is also shorter than the line for the initial *p* of *Pope*.

In French, contrary to the statement of Grammont, the stop for a double consonant is not always held. In fact, in the case of stop consonants, it is never held. (See Figure 10). This is seen both in kymograms and films. In English the stop for a double consonant or for two cognate consonants is usually no longer and may even be shorter (Figures 8 and 9) than the stop for a single consonant. Normally, in English, the stops for a single, double, or for two cognate

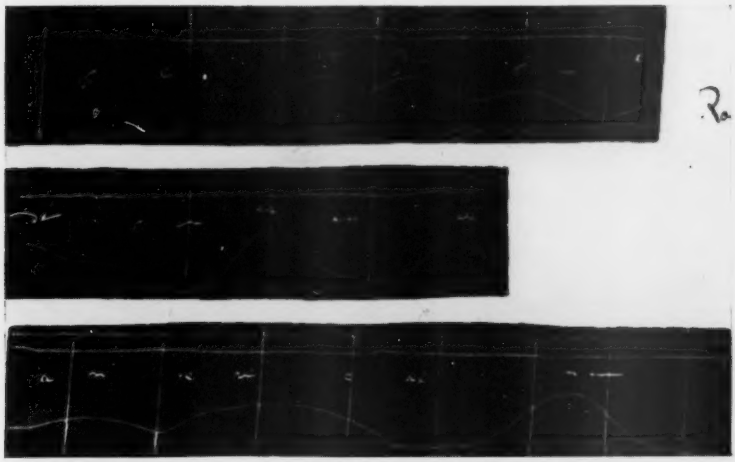

Fig. 10. In the French kymogram of *fête de l'indépendence américaine* the lower line (the line of the tongue) shows separate curves for the *t* of *fête*, and the *d* or *de*, in the pronunciation of the Abbé Rousselot.

consonants seem to be of the same length, and variations, such as they exist, are apparently due to differences in emphasis or of rapidity of speech. This should be the case, because in English, one implosion, one stop, and one explosion serve for a single consonant, for a double consonant, for two cognate consonants, or at times for three cognate consonants, and the same length of time should be required for an identical series of physiological movements.

Explanation of Figure 11

Represents Miss L.'s pronunciation of (*k*)*eep pu*(*tting*) in the sentence: "Keep putting coal on the fire." In frames 1 to 5 the lips are closing gradually for *p*. In frames 6 to 24 the lips are closed for the two *p*'s, the *p* of *keep* and the *p* of *putting*. In frames 25 to 40 the tongue is moving into, through, and out of position for *u* (ʊ), and into position for *t*.

FIGURE 11

FIGURE 11

FIGURE 11

Explanation of Figure 12

(*C*)*oup*(*e*) *plei*(*ne*) pronounced in the sentence: "Oui, oui, elle boit dans une coupe pleine de bière." In frames 1 and 2 the lips are in position for the *u* in *coupe*. In 3 to 7 they are closing for the *p*. In 8 to 22 they are closed for the *p* of *coupe*. In 23 to 29 they are open for the explosion of the *p* in *coupe*. In 30 to 33 they are closing for the *p* in *pleine*. In 34 to 48 they are closed for *p* in *pleine*.

Explanation of Figure 13

(*J*)*ump ba*(*ck*) is being pronounced by an English subject. In frames 1 to 11 approximately, *u* is being pronounced while the lips are closing for the *m* of *jump*. In frames 12 to 40 the lips are closed for the *mpb*. In frames 41 to 48 he is pronouncing the vowel *a* as the lips and jaw gradually open.

FIGURE 12

FIGURE 12

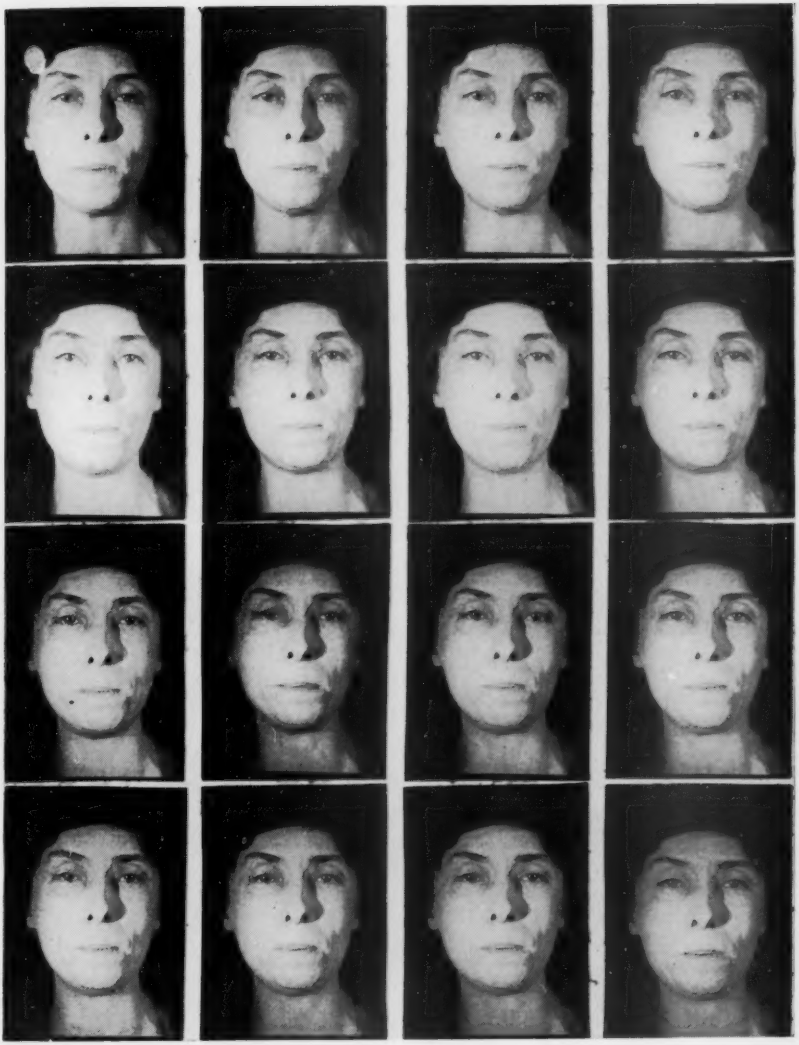

FIGURE 12

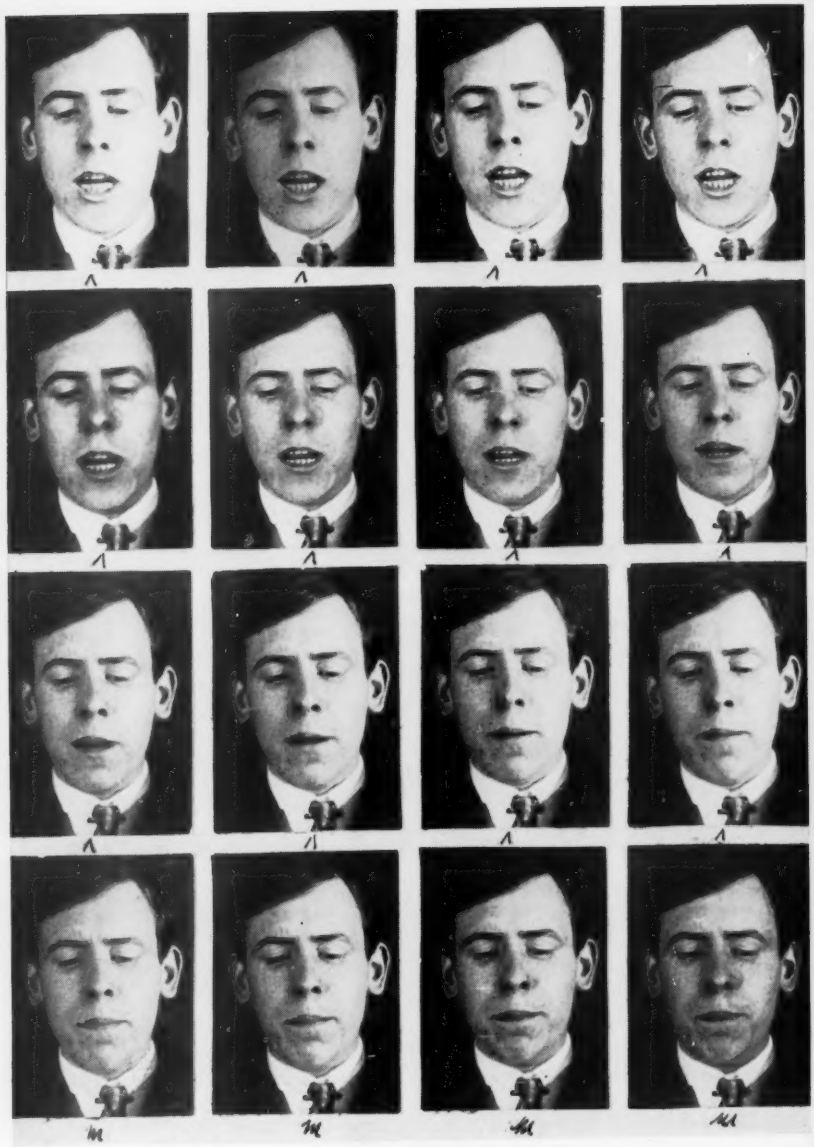

FIGURE 13

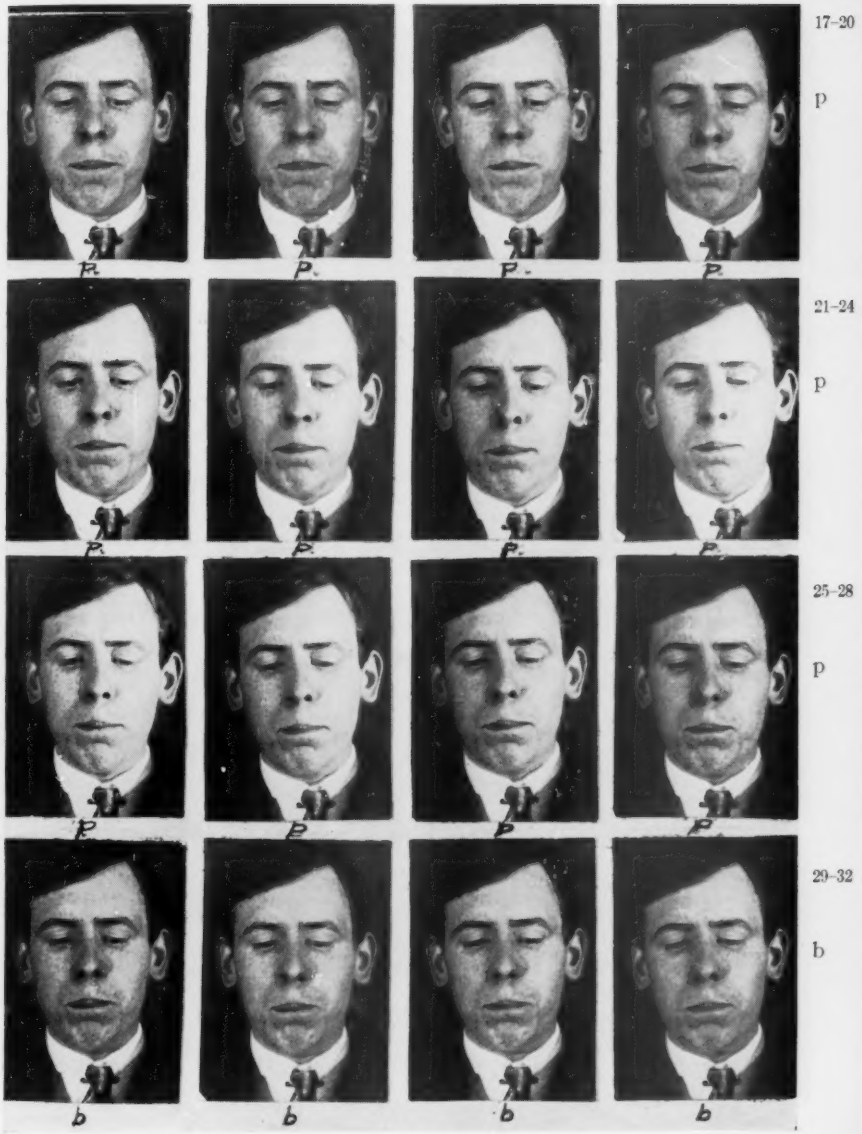

FIGURE 13

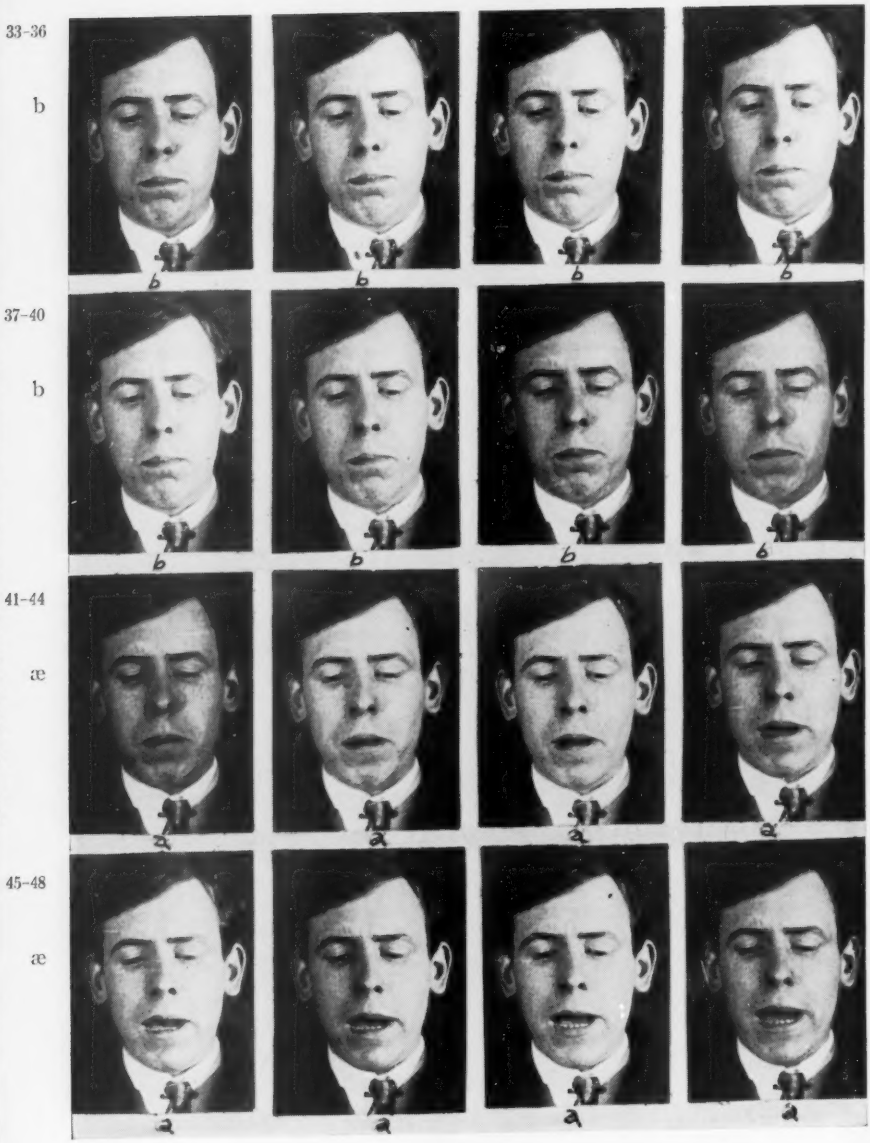

FIGURE 13

Oscillograms also indicate that, in English, there is only one explosion for both consonants, whereas in French, each consonant has a separate explosion.

The dynamic theory of continuous effort from vowel to consonant in English and of interrupted effort to expel the air in French explains the difference in length and voicing of consonants in both languages.

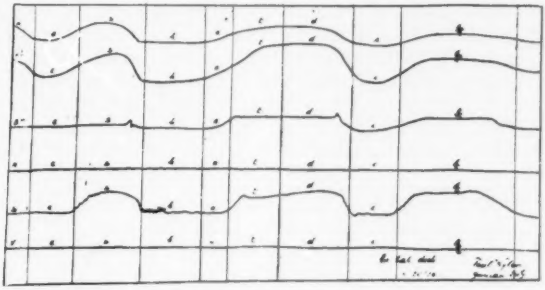

Fig. 14. *Es hat dich.*

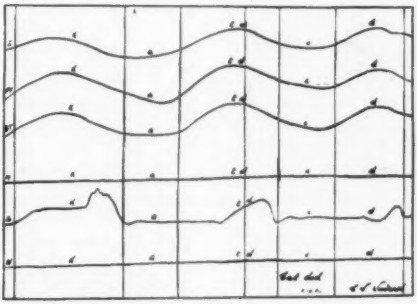

Fig. 15. *Cat did.*

Static phonetics says nothing about this difference in length, and Rousselot, representative of static phonetics, has the following to say in respect to differences in voicing: "A German from central or southern Germany will say, for *bain*, something that will sound to our ears like *pain*. The difference between his *b* and ours comes from the articulation which is stronger with him than with us, and which paralyzes his larynx during the closure of the lips. It is not, however, a *p*, for his larynx vibrates from the first instant of the explosion of the consonant,"[18] and he adds that the German does not succeed in

[18] *Revue des Cours et Conférences*, "Phonétique Expérimentale," M. L'Abbé Rousselot, p. 493. Feb., 1923.

correcting this fault. He also offers no evidence in favor of this theory.

If the static theory of continuous effort to expel the breath in passing in the same syllable from vowel to consonant for German (English), Figures 14 and 15 (Cf. Fig. 16), and the dynamic theory of an interrupted effort for French be assumed to be true, then it becomes apparent that the first part of the stop *td* serves for a voiceless consonant (*t*) and the second part for a voiced consonant (*d*). Consequently, the full stop can be neither voiced nor unvoiced. The time of the stop for the two consonants is, in German as in English, that of a single consonant. On the other hand, in *patte de*, Figure 17,

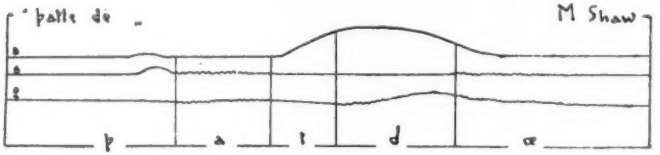

Fig. 16. American pronunciation of *patte de*.

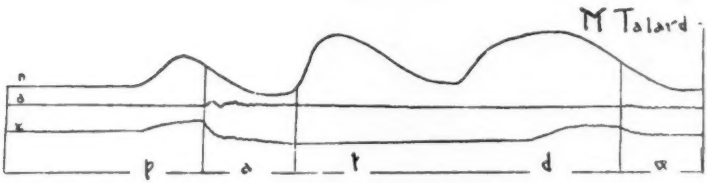

Fig. 17. French pronunciation of *patte de*.

the *t* has its separete explosion, and there are two series of physiological movements—one for each consonant (*t*, *d*)—and it is possible for the first stop to be completely voiceless and the second completely voiced.[19]

Static phonetics observes with Jones that "many foreigners pronounce all the above groups of consonants incorrectly by inserting *h* or *e* between the consonants. The mistake is particularly objectionable in the groups *kt*, *gd*, *act* as *æk^{h}t*, *begged* as *beg^{e}d*."[20] No attempt is made to explain why some foreigners insert the *h* or the *ə* and others do not. If the dynamic theory of an interrupted effort to expel the air between vowel and consonant is true, then this *h* or *ə* is the voiced or voiceless explosion of the preceding consonant.

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[19]James L. Barker, "An Explanation of the Differences in Length and Voicing of Consonants in French and English," *Modern Philology*, Feb., 1929.

[20]Daniel Jones, M. A., An Outline of English Phonetics, p. 144.

The same dynamic explanation holds for the vowel-like sound after *t* in many foreign pronunciations of *it did*. The vowel-like sound is the explosion of the *t*. Similarly the *n* before *t* in the English pronunciation of *maintenant* is explained as a consonant glide between the nasal vowel and the consonant *t*.[21]

The dynamic theory of continuous effort to expel the air within the syllable in English and of interrupted effort to expel the air in the Slav and Romance Languages explains the presence of diphthongs and drawling in English and their absence in French, Spanish, Russian, etc. Since, in English, the pronunciation tends to be continuous, preparation is made constantly one step ahead, and the slow constant shift and continuous effort to expel the air results not only in the diphthongization of both the beginning and end of vowels, but also in the "diphthongization" of consonants.[22]

The interruption of the effort to expel the air while shifting from position to position in the Slav and Romance languages prevents the formation of diphthongs and results in pure vowels and "undiphthongized" consonants.[22]

Dynamic phonetics explains too why Russians and many others are linguistically brilliant in France and rather dull in England. The general mechanism of movement in relation to breath control is the same in Russian and French and different in Russian and English. Consequently, in England a Russian has other, and to him unsuspected, difficulties to overcome.

Are minute differences of position the cause of brogue differences, or are they the result of differences in movement and breath control? For static phonetics they are the cause of brogue differences; for dynamic phonetics they are merely the result of differences in movement and breath control. The tongue touches the palate in pronouncing *tr* in English, Figure 18 and Figure 20, because the continuous pronunciation with uninterrupted effort to expel the air requires a position of the tongue such that *t* may be exploded into the *r*, that is, it requires a compromise position for *t* and *r*. In the Romance languages the *t* and the *r*, Figure 18 and Figure 19, are pronounced with separate explosions, and there is no necessity for a compromise position.

The tongue touches farther forward for *l* in the pronunciation of English *pl* than for *l* in French *pl*, because in English, the *l* is prepared before pronouncing *p*, when the teeth are still close together, and in French the *l* is prepared after the teeth have been separated in the effort to explode the *p* (Figure 1).

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[21]James L. Barker, "Correcting the Mechanism Causing Most Foreign Brogue," *Journal of Speech Disorders*, March, 1936.

[22]James L. Barker, "Rate, Direction, and Continuity of Movement in French and English Speech," *P. M. L. A.*, December, 1930.

The raising of the tongue at the back during the pronunciation of *l*, noted by Jones,[23] results from the merging of the back vowel and *l*; the position for the vowel is held while the position for *l* is being taken.

Fig. 18. English and French *tr*.

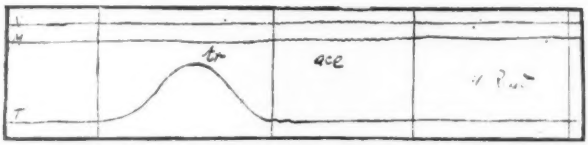

Fig. 19. English *trace*. The *t* is exploded into the *r*, and the pronunciation of both consonants produces a single tongue curve, T.

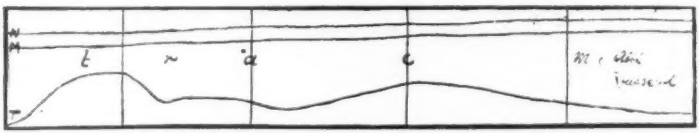

Fig. 20. French *trace*. *t* and *r* are pronounced successively and produce separate curves, the *r* beginning after the *t* is finished.

Positions for the consonants and vowels are compromise positions in English. Few positions are compromise positions in the Romance languages. Anything affecting ease of transition from one sound to another or causing the teeth to be separated at the moment the sound is articulated induces a change in position, a change that is not the cause of acoustic differences, but the result of differences in movement and breath control.[24]

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[23]Daniel Jones, M. A., *An Outline of English Phonetics*, p. 162.

[24]James L. Barker, "Mechanism and Position," *Modern Philology*, November, 1922.

Summary

Phonetics has been based on certain theories; namely, that minute differences of tongue position are chiefly responsible for the differences in pronunciation from language to language, that how these positions are reached and left is of no consequence because the effort to expel the breath is continuous within the syllable, and the shifts in position are predetermined and made in the same manner in all languages.

All the better known phoneticians have based their work on these generally accepted theories, and yet it would seem that these theories of change in position and constant expulsion of the breath are wholly false.

Though it is true that positions are important, there is no one and one only position for any sound, since there are many compensations in position—the tongue does not touch in the same position if the teeth are close together as it does if the teeth are far apart.

English, French, German, Italian, etc., *t*, *d*, *l*, and *n* can be pronounced with any physiologically possible place of tongue-palate contact. Hence, minute differences in tongue position cannot be responsible for brogue differences.

Any comparative experiment shows that transitions from sound to sound are not made in the same way in French and English, German and English, or in English and any other language. Moreover, the way the transition is made is highly characteristic of the language.

Experiments show that the expulsion of the breath is usually continuous in English within the syllable but not continuous in German, French, Italian, Spanish, etc.

For the theory of static positions there should be substituted a theory of position of movement in relation to breath control. Minute differences in position, when they occur, are the effects of the differences in mechanism from language to language and not the cause of the distinctive brogue differences.

The old theory of static phonetics leaves all of the characteristic brogue peculiarities unexplained, such as: in English, slurred vowels, diphthongs, drawling, and in the English pronunciation of French, the presence of nasal consonants after nasal vowels, short and insufficiently voiced consonants; in the English pronunciation of Italian and French failure to pronounce double consonants correctly. In the Romance-Slav pronunciation of English it fails to explain the vowel-like sound after stop consonants, too distinct vowels, lack of proper diphthongs, etc. All known differences are explained and corrected by dynamic phonetics, making use of the theory of position and direction of movement in relation to breath control.

CONVENTION INFORMATION

(See pages 150 and 192 for further information)

ACCOMMODATIONS

College Residence Halls: The Pennsylvania State College can provide dormitory facilities for approximately twelve hundred persons. There are no rooms with private baths. These residence halls are on the campus within five minutes. walk of headquarters and the buildings where the meetings will be held. Charges will be $1.00 per day for each person. Reservations for dormitory accommodations should be sent to Professor R. G. Bernreuter, Psycho-Educational Clinic, State College, Pennsylvania, *well in advance of meeting* dates, giving specific information as to requirements, viz., single room, double room, accommodation for husband and wife, and probable date of arrival and date of leaving. Kindly use the card provided.

Hotels: Good hotel accommodations are available for a limited number. Reservations should be made directly with the managers of these hotels in State College. The hotels and rates are:

	Single		Double	
Hotel	**With bath**	**Without bath**	**With bath**	**Without bath**
Nittany Lion Inn.....	$3.00		$5.00	
State College.....	$2.00–3.00	$1.75	$4.00–6.00	$3.00

Auto Camp: One auto camp, the Autoport, is approximately one and a half miles east of State College on U. S. highway 322. Accommodations include hotel service (linen, cleaning, hot and cold water, showers). Rooms have double or twin beds. Rates begin at $1.50 single or $1.00 double per person for each day.

Meals: Dining services are provided at three convenient centers on the campus, and guests are invited to make full use of these facilities. The first dormitory dining service will open with lunch, Monday, September 2. Regular meals will be served as follows:

Breakfast.....	7:30 to 9:30	35 cents
Lunch.....	11:45 to 1:30	50 cents
Dinner.....	5:30 to 7:00	65 cents

In addition to the residence hall dining rooms, cafeteria service for breakfast and lunch, and a la carte service for dinners will be operating in the Sandwich Shop in the basement of Old Main. This will open for dinner Sunday evening, and be open each day following from 7:30 A. M. to 7:00 P. M. During later evening hours, sandwiches, ice-cream, hot and cold drinks will be available.

RECREATION

The tennis courts of the College will be available and free to those attending the ASCA meetings. The College eighteen-hole golf course adjoining the campus will be open to ASCA members at a special guest fee of 75 cents a day. Special provisions will be made for the entertainment and care of children, including a nursery school for children under the age of four. This service can be more adequately planned if request for it is made early along with request for room reservation.

THE SPEECH DEVELOPMENT OF NORMAL CHILDREN

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This paper stems from a remark made by Wendell Johnson during the discussion following a number of papers on stuttering presented at the American Speech Correction Association Convention, December 28, 1938. Dr. Johnson spoke as follows: "No one can logically lay claim to having discovered the cause of stuttering until he can first predict whether or not any particular young child is going to stutter, before he actually has begun to stutter."

Whether one follows the so-called biological viewpoint on speech defects, or believes that the so-called "psychological" components of social maladjustment, emotional maladjustment, are causative or adheres to the theory of conditioning of inhibition, or accepts the implications of the evidence presented for the cerebral dominant gradient theory, or believes that a semantic problem is largely responsible, it must be accepted that at present no one is able by examining either the anatomical or physiological makeup of any child not stuttering, to predict that this child will stutter. Nor is one able to set up a series of environmental circumstances, such as interference with a definite sided lead, and predict with absolute assurance that the child will stutter. Nor can one set up a series of environmental circumstances dealing with family and home adjustment, emotional adjustments or psychoanalytic adjustments that will produce stuttering in a given child. Nor can one set up an educational or semantic situation that will produce stuttering. The evidence along these lines is clearly conclusive, for many children pass through all of these circumstances so far as we are able to judge, and have no difficulty with speech at all. It is accepted in medicine that in order to prove the cause of a disease it must be shown that individuals with the disease always harbor a particular type of organism, and that injection of this organism into another host always or nearly always produces the disease. In stuttering, and in speech defects in general, this has not been done.

It is true that thyroid medication occasionally produces stuttering, and the author has had the experience of seeing this phenomenon occur. But thyroid only occasionally produces these effects, and thus one cannot predict that giving a child thyroid will produce stuttering.

Further, it has been shown in work from Wisconsin[1] that diabetics rarely stutter. The author has had the experience of seeing stutterers begin to stutter after receiving insulin for diabetes, who had not been known to stutter before administration. But only occasionally does this effect occur. One cannot say that the insulin caused the diabetics to stutter. In fact, the only prediction that can safely be made at this time is that in the insulin treatment of dementia praecox, there is a stuttering phase just before coma, which resenbles true stuttering a great deal.

This paper makes no attempt to solve the problem. It is intended only as a description of the attack that has been begun at the University of Wichita on the problem. The author will welcome the criticism of the members of the Association in the technics and the goal involved.

A few studies have been made on the birth cries of children by means of phonograph recordings. Stinchfield and Young report one such study.[2] There have been a number of studies made by psychologists and others on the normal development of language in the child.[3, 4, 5] These studies have been carried out in the main by individuals unfamiliar with the significance of speech defects, and the data has been assembled by parents, students, and others not even with this small amount of training. The value of these studies is open to question for this reason.

Nor is this the only objection to these studies. Many of them are pure samples from various age levels, taken by different investigators on different children. While not denying that such cross sections are often enlightening, due to the complex environment in which these children live, one cannot say they are controlled. A longitudinal study controlled as far as possible by scientific means has never been made.

The present paper describes the attempt being made at the University of Wichita to answer this need.

While for many reasons it would be desirable to follow the prenatal record of the child, the first contact with the child is made in the hospital. Through the co-operation of the Sedgwick County Medical Society in general, and all of the local obstetricians in particular, a recording apparatus is set up permanently in the

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[1]Schuster, George. Diabetes and Epilepsy Viewed from the Standpoint of Dysphemia. Thesis. 1936.

[2]Stinchfield, Sara M. and Young, Edna Hill. Children with Delayed or Defective Speech. Stanford University Press. 1938.

[3]McCarthy, Dorothea. The Language Development of the Pre-School Child. University of Minnesota Press. 1934.

[4]Shirley, Mary M. The First Two Years. University of Minnesota Press. 1931.

[5]Stoddard, George D., Case, Ida Mae, Wellman, Beth L. and Mengert, Ida G. The Speech Sounds of Young Children. University Press, Iowa City. 1931.

delivery room of one of the local hospitals. As soon as possible after the birth of the child, the attending nurse records his cries. The time of recording of the cry varies from five minutes to half an hour after the delivery. Thus, while it cannot be said that the actual first cry is recorded, still one is unable to detect subjectively any difference between the first cry and the ones following immediately.

This recording is obtained with the consent of the parents, and is requested of them by the obstetrician. At the same time, hospital records are made available, and the obstetrician, if he wishes, adds his comments to the history of the case.

At intervals throughout the week to two weeks stay in the hospital further recordings are made.

The consent of the parents is next obtained to further recordings, and at regular intervals, the child is recorded for purposes of study.

A case history is taken as soon as possible by a member of the University staff. This history records the hereditary characteristics of the family, the presence of various types of speech defectives in the family, history of left-handedness, etc. It records the pre-natal history for illness, shocks, accidents, emotional strains. It studies the occupational level of the family, and the family itself. It records as the child grows older his illnesses, accidents, frights, emotional upsets, etc. The mother also adds her observations, and notes any differences at all in the child's speech. Besides the recording the staff notes these changes and describes them.

In the meantime, the co-operation of the family physician has been obtained, and his observations are added to the record.

Although the project has only been under way since February, a number of significant items have been observed. It is fortunate that recordings have already been secured on children diagnosed as damaged at birth or prenatally by the attending physician, and the characteristics of their cries are clearly and distinctly different from the remainder of the cases in pitch, rhythm, and accentual character, as well as volume.

It is planned to study at least three hundred normal children for at least seven years. Thus the undertaking is extremely ambitious, and may well occupy the attention of the staff for many years to come. Many of the original cases, possibly over half of them, will disappear by moving to other regions, and for other reasons. However, the portion of their life that is caught should prove to be of large value.

Technics of analysis have already begun. The records are being analyzed for pitch, rhythm, accentual characteristics, and roughly for volume. As the child grows older, and begins to develop new sounds, these will be carefully noted. The study is still too new to show anything in this regard.

To return to the introductory remarks. While it may not be possible to predict stuttering, or other speech defects, even after the

completion of this undertaking, still it is obvious that out of this large group, a number of the children will develop speech defects. Here for the first time will be a carefully controlled record of this speech defective child before the speech defect occurs, and it should show the beginning of the speech defect and at least the environmental complexities which have occurred prior to its development. Further, it is well within the realms of possibility from evidence already presented by many that significant differences in the items suggested will appear at a very early age. It then follows that a recording at this age would enable one to predict the development of a speech defect in that child, and it will then be possible to examine this child from all points of view to discover what forces are working or have worked to produce the speech defect.

It also is possible that some speech defects arise rapidly, with no forewarning in the early history of the child. These records should show this and give valuable clues to the unravelling of a number of perplexing problems.

The validity of the case record in use at the University is now being challenged at the University by a controlled experiment on over a thousand cases, and perhaps two or three thousand. It is hoped that this material will be in shape for presentation at the next convention.

The staff wishes to extend to the members of the Association an invitation to participate in the experiment, for the more cases studied the better, and it is hoped that those with facilities for the experiment will join in making it as valid as possible.

COME TO THE CONVENTION

September 2–3–4, 1940

at Pennsylvania State College

A QUANTITATIVE STUDY OF EXTENSIONAL MEANING WITH SPECIAL REFERENCE TO STUTTERING

CURTIS TUTHILL
Temple University

The present study has to do with certain problems relating to stuttering, but uses a technique other than that in the average psychological experiment—the methods and assumptions involved might better be related to what might be called "applied logic," or an experimental approach to semantics.

The problem has to do not so much with stuttering itself, but with the linguistic behavior involved in referring to stuttering either by the stutterer himself or by the non-stutterer. In the usual study, the meaning of the word "stuttering" is either assumed, or is given verbally (i. e., stated to be equivalent to certain descriptive terms whose meaning is assumed). This type of definition, with word related to word, has been termed the "intensional" definition. It is the usual meaning of "definition."

To the logician, on the other hand, the meaning of a word may be thought of *directly* in terms of a set of *objects* to which it refers. The "extensional" definition would be coextensive with the *range* of objects included under the word. It would consist, not of one example, but of the totality of examples to which the word is applied. Obtaining an extensional definition of stuttering would then involve asking a person to point to certain specific examples of stuttering (the opposite, "non-stuttering," might be thought of as being defined, in a sense, by omission). An extensional definition would not involve asking a person what he meant by the word "stuttering" or what he thought "stuttering" to be, but by making a direct word-fact relationship. The present study is interested in seeing to what extent the extensional definition of stuttering would differ with different groups of individuals, chiefly stutterers, non-stuttering "lay" individuals, and non-stuttering clinicians.

The preliminary findings in this study were reported last year in a paper read before this Association by Wendell Johnson. It is the purpose of the present report to indicate the implication of some of the findings to research, diagnosis and therapy. The means of obtaining the extensional definitions consisted of having the subjects listen to a phonograph record, follow along on a transcript, and indicate on the transcript each place where "stuttering" occurred. To measure reliability, or consistency, each subject was asked to repeat his performance a week later.

The sample of speech used on the phonograph record had a large number of "borderline cases" (e. g., slight hesitations, minor breaks in rhythm, pauses in extemporaneous speech, etc.) as well as a number of speech blockages which, because of their severity, would "unquestionably" be considered "stuttering." The particular sample of speech on this phonograph may hardly be considered as an ideal "sample" in the statistical sense. Hence, the absolute amount of disagreement found in this study might be considerably different if a different sample of speech were used. However, it is reasonable to assume that the relative amount of disagreement found among the various groups of subjects would remain the same. For purposes of this discussion, therefore, the findings are indicated only in their topological relationships rather than in their absolute metrical relationships.

As to the total range of items included under the word "stuttering," it was found that the stutterers indicated significantly more items than did the non-stuttering "lay" individuals. The extensional definitions of stuttering were thus significantly different, i. e., not congruent. (The reliability of marking items was not significantly different for the two groups.) This difference in definition has significance depending upon what frame of reference we use. In an absolute sense, we are not justified in saying that one group was "right" and the other "wrong" since at the present time we hardly have an adequate operational definition of stuttering (in terms of objective physiological criteria) to use as a reference point. In the past, most research studies have defined stuttering in terms of subject judgment, and have tried to find physical correlates. That is, they have tacitly assumed the validity of the extensional definition of the subject.

If we take the stutterer as the center of the frame of reference, the definition of stuttering given by the non-stutterers would be "wrong." However, there is a certain justification in reversing the frame of reference and suggesting that, for purposes of therapy, the stutterers' definition *ought* to correspond to that of the non-stutterer; i. e., the stutterers' definition includes too large a range of speech items. The reason for this is that the stutterer's emotional reaction to his speech is made in terms of what he considers the "definition" (non-stutterer's) of stuttering held by the total population, especially the 99 per cent who are non-stutterers.

We may consider the borderline cases which are included in the stutterer's definition and excluded in the non-stutterer's. In reacting emotionally to items of this type, the stutterer is reacting to a type of speech considered "non-stuttering" by 99 per cent of the population. This is a situation which is found in speech therapy quite often, and it is difficult to convince the stutterer that certain breaks in his speech are not heard as stuttering by the hearers; there is a great unwillingness on the part of the stutterer to accept this as a fact.

The range of items indicated by the speech clinicians was not significantly different (numerically) from that indicated by the stutterers. This fact, however, does not invalidate the implication for therapy indicated above, nor the validity of the layman's definition. The social criterion used emphasizes the opinion of the 99 per cent as against the one per cent in the *labelling* of certain speech items.

The reliability of marking items was approximately the same for the three groups of subjects. Several methods of measuring "reliability" for this type of data were contrasted: the coefficient of correlation (number of items common to the two days divided by the square root of the product of the number of items indicated each of the two days), the number of common items divided by the total number of items, and the smaller number of items (one of the two days) divided by the larger number of items. The last method is that which has usually been used in the past to indicate "reliability of subject checking items." When the method of correlation is contrasted with this method, it is evident that the traditional method has probably given a spuriously high picture of reliability.

The variability in the number of items checked by different subjects, even those well trained in speech pathology, would tend to indicate that the "absolute" number of stutterings presented in past studies in the field would probably vary considerably from experimenter to experimenter.

The findings in this study would tend to indicate that, to a certain extent, the stutterer need not consider himself sharply differentiated from the normal speaker, especially if the larger part of his speech breaks are of the "borderline" type. At least from the standpoint of what the average person hears, he need not consider himself sharply marked off from the average speaker. To another stutterer certain breaks may be considered "pathological," but to the average, normal, disinterested listener, they may pass unnoticed. The results in this study tend to indicate, in a number of different ways, that "stuttering" is not a single-valued word with the extension sharply differentiated from that of "non-stuttering." There is wide variability in the way people use the word, ranging all the way from noticeably severe breaks in rhythm to very minor hesitations. When normal extemporaneous speech is embedded in other speech which has more marked fluency ("stuttering") the pauses and hesitations of the "normal" speaker will very often be taken for "stuttering," or at least so labelled. Borderline speech phenomena constitutes a major problem not only in research and diagnosis but in speech therapy and our general orientation with regard to stuttering.

CONVENTION INFORMATION

(*See pages 150 and 184 for further information*)

EXHIBITS

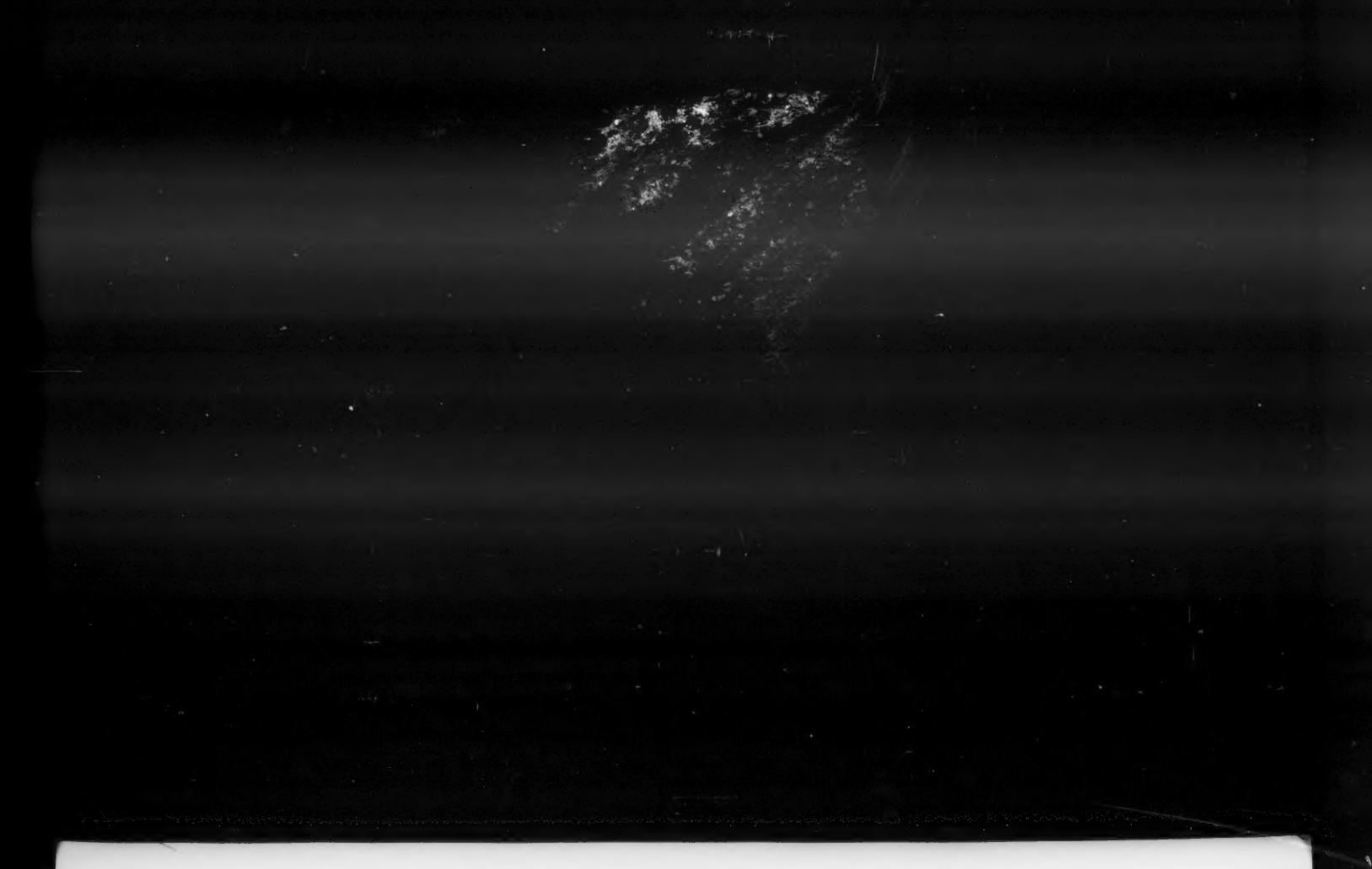
In co-operation with the psychological groups, the Committee on Local Arrangements is planning an extensive display of various types of equipment and books. Audiometers and hearing aids will be a featured portion of the exhibit. ASCA members are encouraged to exhibit unusual laboratory equipment as well as new devices and materials for speech training. Will those who are interested communicate with the Committee on Local Arrangements.

TRANSPORTATION

Highways: Entering from the east is U. S. highway 322, which connects at Lewistown with U. S. highway 22 from Harrisburg and points farther east. U. S. 322 is also the route from Cleveland and the northwest. It makes junction at Port Matilda with U. S. 220 from Altoona and routes from the west and southwest. Pennsylvania highway 45 passes through State College from the north and the south.

Railroads: Railroad stations for State College are on the Pennsylvania lines at Lewistown, 30 miles; Tyrone, 28 miles; and Bellefonte, 12 miles. Those coming from the east should get off at Lewistown; from the west at Tyrone; and from the north via Buffalo and Williamsport, get off at Bellefonte. Latest train and connecting bus schedules are as follows:

	Railroad	**Bus**	**Bus**
Station	Arrives	Leaves	Arrives State College
Lewistown	12:30 P. M.	12:30 P. M.	1:30 P. M.
(from east)	7:24 P. M.	7:24 P. M.	8:25 P. M.
Tyrone	10:28 A. M.	11:56 A. M.	12:44 P. M.
(from west)	12:17 P. M.	1:12 P. M.	2:00 P. M.
	3:03 P. M.	5:41 P. M.	6:29 P. M.
Bellefonte (from north)	3:10 P. M.	3:15 P. M.	3:40 P. M.

Bus Lines: State College is served by the Greyhound interstate bus lines which provide local and express service from New York, Philadelphia, Harrisburg, Scranton, Pittsburgh, and Cleveland. Local bus companies operate between Lewistown and State College and between Bellefonte and State College.

Airways: The nearest transcontinental airway ports are Pittsburgh on the west and Harrisburgh on the southeast, which are on the TWA lines. A plane from the State College airport may be chartered to meet any planes at these or other airports.